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A MANUAL

OF THE

DISEASES OF INDIA

WITH

A COMPENDIUM OF DISEASES GENERALLY

BY

W. J. MOORE, C.I.E.

SURGEON GENERAL WITH THE GOVERNMENT OF BOMBAY
HONORARY SURGEON TO THE VICEROY OF INDIA
PREVIOUSLY FIVE YEARS DEPUTY SURGEON GENERAL HER MAJESTY'S FORCES, PRESIDENCY DIVISION OF THE ARMY; DEAN OF THE FACULTY, UNIVERSITY OF BOMBAY, 1882; PRESIDENT BOMBAY MEDICAL AND PHYSICAL SOCIETY, 1881; FOR SOME YEARS SURGEON TO THE RAJPOOTANA POLITICAL AGENCY, AND SUPERINTENDENT GENERAL OF DISPENSARIES AND VACCINATION IN RAJPOOTANA; FORMERLY THREE YEARS RESIDENT SURGEON AT THE QUEEN'S HOSPITAL, BIRMINGHAM; L.R.C.P. EDIN.; M.R.C.S. ENG.; L.S.A. LOND.

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PREFACE.

SEVERAL YEARS having elapsed since the publication of the first edition of this Manual, it has been necessary to re-write nearly the whole. Some time has also passed since the publication of any work specially devoted to Indian diseases. But recent books on medicine contain chapters treating on tropical maladies much more fully than is to be found in former volumes; and several celebrated Indian medical officers have published important monographs on particular diseases, or on classes of diseases. This modest Manual cannot supersede the study of such volumes. But, as remarked in the preface to the first edition, it is adapted to the trunk, the cabin, the tent, and the march; and may possibly prove welcome when more bulky works, or when volumes devoted to particular maladies, are not at hand. Under this impression a 'Compendium' of diseases generally has been appended.

I do not expect to escape criticism on certain opinions

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expressed with regard to various theories yet finding general support, principally from being taught to successive generations of students. I am, however, content to rest on the old familiar adage, that truth is great and will prevail. And as the expression of the views referred to does not interfere with the more important matters of delineation, diagnosis, and treatment of disease, I have not hesitated to advance them.

Lastly, should this edition be as well appreciated as the first, it will meet what was then, and is believed to be now, a want.

W. J. Moore.

September 1886.

DISEASES OF INDIA.

CHAPTER I.

INTRODUCTORY.

During the first sixty years of the present century the mortality among European soldiers in India[1] averaged 69 per 1,000 annually, while invaliding, during at least the latter part of the period referred to, averaged 29 per 1,000.[2] Surgeon (now Deputy Surgeon-General) Ewart,[3] by independent statistics, demonstrated that the European army in India had hitherto *disappeared* in about thirteen and a half years. Surgeon (now Deputy Surgeon-General) Chevers,[4] comparing other death-rates as given by Dr. Guy, found that while European soldiers in India died at the rate of 69 per 1,000, the mortality among the metropolitan police was only 7 per *mille*. Another writer[5] forcibly remarked, 'At recent periods European regiments in India have melted away like the spectres of a dream. A thousand strong men formed

[1] Sir A. Tulloch's *Statistics. Report of the Sanitary Commission on the Army in India.*

[2] Col. Sykes' *Tables.*

[3] *Vital Statistics of the Anglo-Indian Army.*

[4] *Ind. An. Med. Sc.*, 1860.

[5] 'The British Juggernaut in India.' *Sanitary Review*, Oct. 1857.

this year a regiment. A year passed away, and 125 recruits were required to fill the broken column. Eight years passed away, and not a man of the original thousand remained in that dissolving corps.' And Colonel Hodson[1] asserted that the British soldiers who then served in Bengal one year encountered as much risk of life as in three such battles as Waterloo. I calculated[2] that by this mortality and invaliding two millions of money were expended, or more than one-fifteenth of the then revenue of British India. At the same time, as nearly as could be ascertained, European soldiers' wives died at the rate of 35½ per 1,000 annually—the home death-ratio of women at the same ages being, according to the Registrar-General's report for 1854, only 11·96 per *mille*. European soldiers' children died in Bengal at the rate of 84 per 1,000—the English rates in twenty-four large towns being, at the same ages, 2·36 per *mille*.

Among the sepoys composing the native army the mortality was also undoubtedly high, but no reliable statistics are forthcoming showing the exact death-rate which prevailed.

Similarly the general civil population were known to furnish a high death-rate—in some districts, as it was presumed, to the extent of 40 or even 50 per 1,000 annually. But as there were no attempts to secure vital statistics, it was impossible to ascertain the mortality with any approach to accuracy.

Having these facts in view, and seeing in the diseases of both Europeans and natives the evidence of preventable malady, a band of sanitary pioneers arose in India, many of whom have long since joined that spirit-world of which although so near we know so little. I, as a humble member

[1] *Military Miscellany.*

[2] The Author's *Health in the Tropics; or, Sanitary Art applied to Europeans in India*, 1862.

of this sanitary pioneering band, joined in reiterating in various places,[1] and notably in the former edition of this manual, the means which should be taken to render India more healthy for natives, and more habitable for Europeans. It was pointed out that although sanitary arrangements, begun at home, had been extended with success, especially among the troops, to the Canadian stations, to the Mediterranean commands, to the West Indies, &c., India, with its then 80,000 European soldiers and vast population, remained to be dealt with. It was not, however, until the late Lord Herbert, then Mr. Sidney Herbert, with Crimean recollections and experience fresh in memory, turned his attention to Indian sanitary matters that Parliament were impressed with the necessity of action. And to this end the powerful aid of Miss Nightingale was effectually rendered. Hence the formation of the 'Royal Sanitary Commission, appointed to enquire into the sanitary state of the Army in India,' whose report was published early in 1863. The small amount of attention previously vouchsafed to Indian sanitary matters was painfully exemplified by what followed the publication of this report. The announcement made by the Commission, that since the commencement up to the termination of the first half of the century the rate of mortality among Europeans in India had averaged 69 per 1,000 annually, was received with astonishment and indignation. Sir Charles Wood, then Secretary of State for India, made in the House on July 14, 1863, the extraordinary assertion that 'the report of the Commission had brought to light a rate of mortality which before its publication no one believed to exist.' The *Times* remarked, 'People might be prepared to hear the mortality was double that which prevailed in English barracks before anything was done to improve them,

[1] The Author's *Health in the Tropics; or, Sanitary Art applied to Europeans in India.*

but even this exaggerated estimate would fall far short of the truth.' And other journals, even some medical journals, followed suit, commenting on the *discovery* of the great mortality of Europeans in India. But at the very time the House of Commons listened to the assertion of the Secretary of State for India, that the Sanitary Commission had *discovered* a rate of mortality which no one had believed to exist, such works as Colonel Sykes' 'Statistical Tables,' Macpherson's 'Statistics,' Ewart's 'Vital Statistics of the Indian Armies,' and 'Vital Statistics of Indian Jails,' Chevers' 'On the Means of Preserving the Health of the European Soldiers in India,' my own 'Health in the Tropics, or Sanitary Art applied to Europeans in India,' had long passed through the press, all reiterating the oft-told tale of men *disappearing* at the rate of 69 per 1,000.

Yet notwithstanding this, the few survivors of the original band of sanitary reformers have had the satisfaction of seeing almost every recommendation, collectively or individually made, and so long ignored, at last carried out. Thus the improvement of the public health of the natives generally had ever been held as one of the most important measures by which the health of the Europeans sojourning in their midst was to be maintained; and the initiatory step was taken immediately after the publication of the report of the Royal Sanitary Commission, by the formation of sanitary boards in each of the three Presidencies, who were 'not to be merely deliberative bodies,' but were 'to exercise a proper supervision over the whole sanitary administration.' It, however, soon became evident that boards composed of four members and a secretary were too cumbrous and expensive, so boards were replaced by one commissioner and a secretary, the latter being a medical officer. This arrangement also proved a failure. Sanitation may, of course, be separated from the practice of physic—perhaps should be so—but it cannot be isolated from an acquaintance with the etiology

and causation of disease, from physiology, chemistry, and medical climatology.

Sanitation could not succeed when the sanitary commissioner was dependent on his secretary for even the meaning of ordinary medical terms, and so soon the commissioners retired, and the medical secretaries became the commissioners. Had medical officers been utilised from the first, it might not have been considered necessary to submit a code of sanitary regulations for India for home revision. But it was remarked in an official resolution, 'Many officers serving on the Indian Sanitary Commissions will necessarily be unacquainted with the more recent improvements in sanitary science.' Allowing that as the boards were constituted such was the case, it may be confidently affirmed that neither now nor in 1868 was there any lack of officers in India quite as capable as any at home to carry out Indian sanitary reforms. Otherwise the services had not possessed in each Presidency the various excellently and specially qualified officers who might be named.

Next we have the establishment in all towns, and in many villages, of either municipalities or sanitary boards who, under the guidance of the civil district authority, are presumed to attend to all matters of local sanitation as advised by the Sanitary Commissioner and his deputies. The sanitary authorities are now also in most districts entrusted with vaccination, which during recent years has made much progress in India. Similarly, the provision of medical aid for the people has so increased that there are few districts or even large towns in which there is not a hospital or dispensary, now probably to be supplemented by special institutions for females. Then there is the introduction into many towns (especially in the Western Presidency, where the physical features of the land are more favourable) of water from the distant hills, resulting in the diminution of disease, as in Bombay for example, where guinea-worm,

formerly so prevalent when drinking water was obtained from local wells, is now seldom seen. Next we have the adoption of an extensive system of forest conservancy, on which the equalisation of the rainfall and moisture so much depends. Neither must the renovation and extension of irrigation works be forgotten, by which millions of acres once sterile and unproductive have been brought under cultivation. For although over-irrigation and profuse waste of water have been proved to maintain the water in the wells at a higher elevation, to render the ground damp, and so to excite disease, this cannot be accepted as a reason against irrigation properly controlled, any more than accident or carelessness can be regarded as reasons against railway extension, which extension combined with irrigation may be regarded as double security against future Indian famines. The cheapening of that important item of consumption, salt, consequent on the assumption by the British Government from the native states of the great salt-producing districts of Rajpootana, must also be regarded as an important sanitary advance; salt being a necessity in the animal economy, from the want of which natives in many districts have suffered greatly. The successful introduction and growth of cinchona in India, followed by a supply of cheap cinchona alkaloids, is another important sanitary step long since advocated by medical officers. In order to promote sanitary knowledge among the natives numerous vernacular pamphlets have been published, the Government of India offering a prize for the best essay of the kind. For Europeans there is my 'Manual of Family Medicine for India,' and it might be added of personal hygiene, which also had its origin in a similar offer made by Government, and which has now, by the desire of Government, gone through four editions.

Next, there is the Countess of Dufferin's Fund, and the National Association for the medical tuition of women

for the medical relief of women, and for the supply of trained female nurses and midwives, from which much benefit may be expected to that extensive class of females, principally belonging to the higher orders of society, who are not permitted to appear in public or even to see a male doctor. Lastly, in all provinces there is a more or less accurate system for the registration of vital statistics, which in some degree at least renders it possible to judge of the benefits to public health resulting from all that has been effected.

Turning from civil to military matters, we find that, as long previously suggested by medical officers, more attention than formerly is now given to the selection of recruits sent out to India. They are not, or at least it is ordered they should not be, sent out so young as previously, that they should learn their drill in a temperate climate before embarking, and that they should arrive in India during the cold season. Better means of transport were provided in the magnificent troopships which have now long safely and expeditiously voyaged between England and India. Much money has also been spent in the construction of barracks for the Anglo-Indian army, giving abundant cubic space, and in which all necessary appliances for coolness and comfort are allowed. Similar remarks apply to hospital accommodation. The dietary has also received much attention, resulting in an increase in the proportion of anti-scorbutic articles of food. The deleterious clothing of former days—the tight stock and the coatee—has been replaced by a more rational dress, and the texture of the dress is now regulated according to the season instead of being left to the caprice of any one person. Cantonment or sanitary committees are in working order at every military station, whose duty it is to supervise the local sanitary arrangements, and to maintain the conservancy, drainage, and general cleanliness of the locality. The employment and amusement of the troops have also received much attention, so that no soldier need

now, from sheer ennui, sleep his idle hours away; the cause, as Macnamara years ago pointed out, of fatty degeneration and hepatic disease. The prevention of venereal has been sought by the registration and examination of females, and by the institution of lock hospitals. The more extensive utilisation of the hill ranges of India, not only as sanitary stations, but also for the permanent cantonment of European troops, has also made some progress, although not to the extent which could be wished. Invaliding is now effected so that the sick have a fair chance of recovery of health, soldiers not being kept in the country, as formerly, until their condition was hopeless. Also, as Dr. Ewart [1] has demonstrated, the discarding of the spoliative system of medical practice which prevailed even so recently as a quarter of a century ago, and the substitution of a more rational system, in which the bleedings, leechings, purgings, and salivations of former days have no place, has not been without influence on the mortality. Much also must be attributed to better habits of life generally, and especially to the substitution by the upper classes of claret and other light wines or beers, for the heavy malt liquor and alcoholic mixtures formerly so much used; and to the diminution of the amount of spirits formerly obtainable by the soldiers from the canteen.

Lastly, while both Indian civilians and military officers are, in common with the general public, better informed in matters of sanitation and personal hygiene, medical officers pass through, at Netley, special instruction in such subjects, while their rank and status has been increased; so that such a state of matters as once pictured by the late Lord Herbert, by Sir Ranald Martin, and by myself, could scarcely exist. Lord Herbert wrote, 'When a medical officer goes to the general commanding, who under a tropical sun, up a river surrounded by swamps, is feeding his troops with salt pork, and tells him that unless he gives them fresh meat and

[1] *Review of the Treatment of Tropical Disease.*

vegetables they will be down with scurvy and fever, he does no more than his duty. But if he is met by the man in authority by the rejoinder, "Sir, when your advice is wanted it will be asked for," he probably vows never again to expose himself to such a rebuke. Six weeks after he is called upon to cure disease which is not curable at all, though care and attention a few weeks earlier might have obviated much of it.'

Sir Ranald Martin wrote, 'When serving in one of the most pestilential countries known in India, I made a topographic examination of the localities, and reported the result to my commanding officer, suggesting the most suitable arrangement for encamping the men against the coming rainy season, when it was well known that a great increase of deadly fever would result. The answer was, "I'll be d——d if I do!" Now here was no blundering lieutenant, but one of the most able and well-informed field officers I have ever known: yet such was his treatment of a grave matter of duty, the neglect of which before the year was over cost him his life.'

In 1856 I wrote, 'When up the river Euphrates in the Hon. E. I. Company's brig "Tigris," the men were obliged, in consequence of the extreme heat, to sleep on deck, and although the river is lined on either side by miasmatous date-groves and rice-fields, still it was with difficulty I induced the lieutenant in command to protect the men from the heavy dews and floating malaria. Again, when in medical charge of the Hon. Company's steam frigate "Punjaub," first with the Bombay Light Battalion on board, and secondly with the right wing of the 2nd Bombay Europeans (for the theatre of war in Persia), it was only by urgent remonstrance backed by the surgeon in charge (and which remonstrance led to much unfriendly feeling) that I prevailed on the first lieutenant and officer in command to spread the awnings at night over the men sleeping thickly

on deck from the forecastle to the wheel. The answers I frequently received when persisting in nightly recommendations that the awnings should be spread were, that a sudden squall might gather under them; that it interfered with the ventilation of the ship; that it was not man-of-war like; that the excessive dews of night rotted the awnings. Yet the capstan, made of solid wood and brass, was religiously covered every night, while tender flesh and blood—men going to fight their country's battles—might be exposed to the destruction of their health and the impoverishment of the State; executive officers of those days holding the idea, as was indeed more than hinted to me, that the medical officer's duties should be confined to feeling the pulses and examining the tongues of his patients.'

Times, however, are now happily changed. Both civil and military authorities listen willingly to any reasonable suggestion of the medical officer, and are ready to carry out any practicable recommendation, unless positively prevented by the absence of pecuniary means. It is not, therefore, now necessary in a work of this kind to preface the purely professional part with remarks on the means of preserving health in India as it was judged desirable in former years, when the first edition passed through the press. At the same time the means by which the good results which have been obtained are to be enhanced may be mentioned. These are, as regards the civil populations, a steady persistence in the course now pursued, especially as regards the extension of means of communication, by which scarcity of food in one province while plenty prevails in an adjoining district may be avoided. For it should be regarded as an axiom that a poorly fed population are more liable to any form of disease than a fairly nourished people, and are especially liable to the classes of disease which afford the highest rate of mortality throughout India, viz. choleraic maladies, scurvy, and so-called malarious fevers, and their sequelæ. Without

denying the advantage to public health derived from drainage conservancy and other sanitary measures, I regard the equalisation of the food-supply as of the greatest importance in India, and next to this the abundant supply of pure water. Moreover, the equalisation of the food-supply, and the supply of pure water to many localities, is practicable, while the sanitation of many villages is at present impracticable. Many villages there are, where from defects of site, construction, surrounding jungle, &c., removal and reconstruction is the only effective sanitary measure; others in which the population are too ignorant and too poor to do anything for themselves, and the villages, collections of grass or mud huts, too impermanent to warrant the expenditure of public money on costly sanitary measures. Then there is a considerable nomadic population, towards whom, until their mode of life is altered, sanitation is impossible. Yet, it is the populations composing the two classes last-mentioned who suffer when epidemics are abroad, and whose locality becomes the centre of infection. The subject of village sanitation in India is one of admitted difficulty, and what is now required is a sanitary executive, as well as a sanitary counsellor, by which agency more at least might be effected than is now possible, although all that is desirable might not be practicable.

As regards the European military, soldiers are still sent out too young. The Army Sanitary Commission recommended that no recruits should go to India until 25 years of age, and the nearer this age is approached the more likely will recruits be to maintain a healthy standard. Then, I think, more attention should be paid to the selection of recruits for Eastern service, on which subject some remarks will be found under Anæmia (p. 22). Then more anti-scorbutic vegetables are required in the dietary. More systematic measures against venereal are also desirable. Lastly, a greater utilisation of the hill climates of India, not

only as sanitary stations, but as permanent cantonments for European troops, to which regiments fresh from Europe should be sent preparatory to future service on the plains. That more remains to be done than has been effected is sufficiently evident from the one fact that the reduction in the mortality of soldiers' wives and children has not kept pace with that of the men.

As regards the native military force, the principal requirements are better dwellings than so many of the 'lines' on which the men live now are; more systematic drainage of the immediate locality; permanent increase of pay in those localities where food is extraordinarily dear; and a system of retirement not entailing the necessity of an invaliding board reporting the man to be quite unfit for further service.

By such means the value of native civil life will be further reduced from the 24·30 per 1,000, to which (according to the last statistics available for the whole of British India) it has been lowered from an unknown quantity. The value of European military life, which during the last twenty years has been annually increasing gradually (except in 1879–80, during the Afghan war), will be further permanently enhanced to the 12·56 per 1,000 to which it once attained (in 1884). And as regards civil European life, we may see all insurance offices, instead of some companies, offering policies at European rates. Lastly, the native army may become what Dr. Fraser years ago asserted it ought to be, viz., the healthiest in the world, and the only military force wherein the mortality at the same ages is not greater than that of the population from which it is drawn. From the most recent statistics the death-rate of the native troops is scarcely 11 per *mille*, and there is no doubt that this may be further reduced among a body of carefully selected young men such as should compose the sepoy army.

The death-rate of some Indian jails is still a blot on

sanitary administration, although the total mortality in jails, viz. 29·11 per 1,000 in 1884, when compared with the death-rates of the outside population, is not excessive. For prisoners are subject to special influences, and the mortality is small as compared with that of former years, or with that obtaining in the prisons of the Native States. But in the absence of cholera, in one jail in 1884 the mortality amounted to 183 per *mille*; in another to 136; and in a third to 76. These facts require that such extreme cases should be taken in hand, and the causes, whatever they may be, traced and rectified, even if entailing the construction of new buildings.

It is not, however, to be supposed that a tropical country such as India can ever be so conducive to longevity, either to Europeans or natives, as a temperate climate is, at least, to the former. Europeans must always pay a certain penalty for the change of climate and circumstances involved by a residence in the East. And it has been sufficiently proved that natives of India, even of the higher classes, exhibit marks of senility, and die at an earlier age than Europeans. Thus, Dr. Maconachie, ophthalmic surgeon, Bombay, has recently stated that cataract—an acknowledged mark of senility—occurs at a much earlier age among natives than Europeans. And it has been shown that the average number living at each age-period in the Bombay Presidency, as compared with England, is, after the age of fifty, altogether in favour of England. In Bombay the percentage from fifty to fifty-nine is, of men 5·85, of women 6·32; in England it is 7·25 and 7·38 respectively. In Bombay the percentage from sixty years and upwards is—men 3·99, women 5·07; in England 7·10 and 7·83 respectively. Still, as previously remarked, much has been done and more will be done to render India a healthier country for both Europeans and natives. Dr. Fyrer, who visited Bombay in 1672, said of the Europeans there, 'I reckon they walk in charnel-houses;

in 500 one hundred survive not.' Now Bombay, with its water-supply (still to be augmented), with its extensive system of drainage and sewerage in progress (regarding which, however, some difference of opinion exists), and with its able health officer is, perhaps, the most salubrious of tropical cities.

CHAPTER II.

ANÆMIA.

Anæmia really signifies lack of blood, while its synonym, *Oligæmia*, signifies deficiency and poorness of blood. The terms *Oligocythæmia* and *Hypalbuminosis* are also used, the first as indicating a deficiency of red corpuscles, the second a deficiency of albuminous constituents. The term oligæmia perhaps comprises the exact condition better than any other in use, for there is not only anæmia or lack of blood, but there is also a poorness of blood, consequent on a diminution in both quantity and quality of certain constituents; characterised by peculiar pallor, impaired nutrition, and general debility—a condition which appears in all shades and degrees, and which, while constituting a serious malady *per se*, is also the foundation, by the slow blight it effects of the constitutional powers, of many of the diseases of tropical climates.

Anæmia is to be traced to a disturbance of healthy nutrition, consequent on a want of due proportion between the consumption and renewal of functionally important elements of the blood; and this disturbance of nutrition may be brought about, not only by too little matter for consumption or renewal being offered (as when anæmia results from starvation), but also by improper material being offered (exemplified by scorbutic anæmia), and by error or inability on the part of the renewal agency (instanced by anæmia from indigestion and spleen-disease). In addition, anæmia

may arise from abnormal acceleration of consumption (as from hæmorrhages and exhausting discharges). Anæmia may also be caused by intestinal parasites, especially by tape-worm. Lastly, several of these conditions may exist together, each exerting its separate influence in the production of anæmia.

Blood-changes of Anæmia.—A thousand parts of blood contain nearly eight hundred parts of water, the red corpuscles, albumen salts, and fibrine making the sum total. While the albuminoid compounds supply the pabulum for the nourishment of the tissues, the red particles, by conveying oxygen and removing carbon, are instrumental in effecting changes without which such nourishment could not progress. Although it is probable that the remaining constituents of the blood, the salts and the fibrine, may undergo some alteration, the present state of our knowledge of the subject leads to the belief that anæmia must be ascribed to a deteriorated condition of albuminoid compounds and of red particles. In what precise manner the former are deteriorated cannot be even safely conjectured; but it may be surmised that the power of corpuscle-formation is more or less abolished, and that corpuscles formed have little power to absorb hæmoglobin, and consequently do not reach their full development.

Anæmia is usually divided into *idiopathic* or *primary*, and *symptomatic* or *secondary*. Idiopathic anæmia appears, however, a misnomer, as the malady must always be symptomatic of some deteriorated condition, although it may not be easy to discern what the deteriorated condition really is. Thus so-called idiopathic anæmia may be developed by want or poor or insufficient diet, not amounting to actual starvation; or by diet deficient in some necessary element. Whenever famine has occurred in India large numbers of the population, suffering from scarcity but not actually famine-stricken, have become anæmic, or scorbutic, or both.

There is strong reason to presume that idiopathic anæmia may be developed by want of light and fresh air. But as persons deprived of such essentials are usually subjected to other morbific influences, the actual effects of the former have not been accurately gauged. It is, however, believed that the absence of solar light and fresh air interferes with the formation of red corpuscles; perhaps because less red corpuscles are required when there is less oxygen for them to carry, as would be the case in the absence of fresh air. The bleaching effect of want of solar light on certain vegetables is well known; and there is little doubt that a similar sinister influence is exerted on animal life. So-called idiopathic anæmia may also originate from either excess or deficiency of physical exercise. While excessive fatigue leads to increased consumption of tissue, which if not replaced must terminate in progressive debility, deficiency of exercise or prolonged repose leads to loss of appetite and dyspepsia, and consequently reduced supply of nutrient material. Or in other words, as quicker muscular action short of fatigue leads to accelerated digestion, so the reverse. Other often unrecognised causes of so-termed idiopathic anæmia are venereal excesses and masturbation, both involving the exit from the system of a discharge rich in material. An unsuspected scorbutic or syphilitic taint may also cause anæmia; or the poisons of lead, mercury, iodine, aniline, and tobacco. Then anæmia may possibly arise from depressing emotions, which have been long recognised as rendering the 'cheek grow pale' and 'gnawing at our life and health.' How depressing emotions act in the induction of anæmia is not evident, unless indeed the mental condition, leading to neglect of exercise and consequent loss of appetite, entails the result of diminished nutrition.

The anæmia usually regarded as *symptomatic* arises from a great variety of causes, many involving organic disease. Prolonged lactation is a fertile source, and so are many patho-

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logical discharges, in proportion to the albuminoids they contain. Of these spermatorrhœa, leucorrhœa, gleet, albuminuria, chyluria, and chronic dysentery may be mentioned. Malignant growths, again, produce anæmia by the constant drain they maintain on the nutrient resources of the blood. Symptomatic anæmia may also be the result of certain poisons previously referred to, of animal parasites, of spleen disease, of scurvy, and of venereal. It may also arise from hæmorrhages external or internal. Hæmorrhoids, for instance, are a frequent source of anæmia. After profuse bleeding the blood is not only diminished in quantity, but is relatively poorer in albuminoids and red corpuscles, and richer in water. This is probably explainable by the diminution of blood-pressure which the bleeding produces, and by the consequent dilution of the blood from the tissue juices by watery osmotic current.

In addition to these and other manifold *causes* of anæmia which may excite the malady in any climate, there is in the tropics an additional potent factor in the heat of the climate, and it is therefore in tropical countries that anæmia is most prominent. Apart from special forms of anæmia, not excepting those which are reputed to arise from so-called malarious influences—the *malaria chlorosis* of Vogel, or the *paludal cachexia* of Martin—there remain a very large number of cases in which the poorness of the blood must be attributed to heat, and to which the term tropical anæmia is more correctly applied. It is a mistake to doubt, as some have done, the injurious effects of continued heat on the blood of the European especially. Experience indeed teaches us that extremes of heat and of cold, both, exert a weakening influence on the system, although such influence may be exerted in a different manner; and the directly injurious effects of a high temperature on the blood cannot be ignored. This view is to some extent supported by the fact of languor and tendency to anæmia being frequently observable during

extraordinarily hot summers in temperate climates. Although tropical or heat anæmia simulates other forms of the malady, as for instance the condition arising from so-called malarious influences, or from insufficient exercise, or from want, still tropical anæmia manifests itself in persons who have not suffered from malarious influences, who are obliged to lead an active life, who have not suffered from any kind of want, who have not indeed been subjected to any other cause of the anæmic state than heat. Sir J. Fayrer and Dr. Ewart describe *anémie tropicale* as occurring to those prolonging a residence on the plains, and as being generally preceded by a history of repeated attacks of fever, splenic tumour, and derangement of digestion. But such cases would also probably include the condition known as *leucocythæmia*, in which the further change of white-cell blood appears. I would limit the term tropical anæmia to those instances—and they are numerous enough—in which anæmia presents without any such preceding affections as named above. For anæmia, after such affections, may and does occur,[1] not only in tropical climates, but also in semi-tropical countries, and even in temperate climates; whereas heat anæmia is essentially a tropical malady. Anæmia is indeed the cause of fever, of spleen disease, and of digestive disorders, quite as often as the consequence.

The fact is that *blood-degeneration* is initiated in the system of the European from the very commencement of tropical residence. Brunton has shown that by simply keeping an animal in a hot chamber for a little while, the tissues decompose more rapidly, and evidence of their waste is to be found in an increase of urea in the urine. And so it is with the European transported to the tropics, on whom the long-continued heat acts injuriously in various ways. The greater portion of the waste of the body or of material which,

[1] *Du Traitement des Maladies Tropicales*, 1883. Read at the International Medical Assembly, Brussels.

having served its purpose within the body must be expelled, is passed off by the lungs and liver. The oxygen taken into the lungs unites with the carbon of the blood, which it thus cleanses of noxious or effete matter, returning it to the atmosphere as CO_2. In a temperate climate a full-grown man thus gives off about eight ounces of carbon every twenty-four hours. But the atmosphere of the tropics is, from heat, more rarefied than in a cold climate, the result being that a given bulk of air contains less oxygen in the former climate than in the latter. And although this diminution of oxygen may be slight, it must be recollected that it is continuous; and as half a degree of temperature makes the difference between the fluidity and solidity of water, as a quarter of a grain will turn the balance of a million grains, as an almost imperceptible increase of impurity of air will cause the difference between health and disease, so a very slight diminution of oxygen will lead to inevitably injurious consequences. Ziemssen states, 'The exhausting influence of hot climates is in a great measure to be accounted for by mere bodily inactivity.' This, however, is only half the truth. But as very generally (owing to the small amount of cool suitable time available, and to the lassitude produced by heat), comparatively less exercise is taken in the tropics, it follows that the breathing is less accelerated by motion, resulting in a diminished bulk of air being inspired, and hence again a smaller amount of oxygen. As a necessary consequence of these *two* distinct manners in which the supply of oxygen is curtailed, the carbon breathed out from the lungs in the shape of carbonic acid is diminished in quantity. In other words it may be stated, the higher the temperature and the less the exercise, the less carbon is exhaled from the lungs. With the diminution in the supply of oxygen there is probably a less demand for the carriers of oxygen—the red corpuscles—and hence they begin to decrease in number, or to lose their different functional powers. Neither can such

exercise be taken as would by accelerated respiration, afford the needed oxygen without *fatigue*, probably still more injurious. Also excretion is slow, continuous perspiration leaving scarcely sufficient water to carry off urates. Therefore some organ must perform compensating work, or the blood becomes charged with noxious materials. Again, the air which is expanded by heat is less elastic than cold air, and thus less fit to dilate the lungs and chest. Or, in other words, the pulmonary capacity is smaller, and the pulmonary dephlogistication is slower from continually perspiring in hot air. This leads to less determination of blood towards the chest, to lessened sanguification and warmth of blood, to accumulation of blood in the umbilical region and *vena portarum*; to at first an increased hepatic dephlogistication and augmented secretion of bile (but as hepatic congestion merges into hepatic deposit, the reverse); and to a substantial modification of the blood with noxious materials. The anæmia therefore starts from a disturbance of sanguification, leading to enfeeblement of digestive powers, which still further disturbs sanguification, and thus a vicious circle is established. Doubtless, if persons entering the tropics accommodated their living to the altered circumstances in which they are placed, such results might be to a great extent deferred or prevented. But as a rule such endeavours are not made. People entering India frequently live as before, or even consume more rich food, or, in consequence of thirst, or under a mistaken idea of 'supporting the system,' more ale, wine, or liquor than they had been accustomed to consume in Europe. But the effete matter, not required for the nourishment of the body, must be removed from the system, and so the liver especially is excited to additional action. As a consequence the liver may become congested or even more seriously diseased; or, failing to perform its functions, the bowels may be compelled to compensating action in the form of diarrhœa. But notwithstanding

this, by the retention of carbonaceous matter in the blood, this fluid becomes depraved and deteriorated, and is, in fact, in a semi-poisoned condition, while the red particles still further decrease in number. There is, in fact, that predisposition to disease and especially to zymotic disease established, which Carpenter stated may be generalised under one expression—viz., 'the accumulation of decomposing nitrogenous matter in the blood, and on which pabulum zymotic poisons multiply when they would be harmless in pure blood.' Thus, if the personal hygiene and sanitary surroundings of the individual are not satisfactory, deleterious matters are not only formed within the system as explained above, but they may also be introduced from without, thus adding to the pabulum of disease. Thus the system is predisposed to impressions from external cause, the principle of which is *chill*. If some acute malady, as so-called malarious fever, does not occur, other changes do, as detailed under the symptoms of ordinary anæmia.

It must, however, be noted that during recent years a considerable change for the better has taken place in the habits of Europeans. As noted at p. 8, less fermented liquor, and of a less deleterious character, is consumed, while since the institution of lawn-tennis more exercise is taken. Thus the influences causing anæmia have been reduced in magnitude, although from the carelessness of the parties concerned the danger from 'chill' has been increased.

The *temperaments and constitutions* which are most liable to be affected by the causes of tropical anæmia may now be considered.

In former times much more attention was paid to temperament and constitution than latterly. It was held that peculiar temperaments fitted men for different positions in life, and I am not sure that this wisdom of the ancients has not been too much ignored by the moderns. When the Aryans first descended from their northern homes, and

dividing into several great streams invaded new countries and founded new empires, there is reason to believe their complexions were white, and hence that their temperaments, and constitutions, and habits (for we know from the Vedas they were meat-eaters) were those of, or nearly approached those of, northern European races. The Aryan western offshoot, which afterwards became the Germanic stock, were certainly white, or climate and circumstances have made them so. And we have the fact that the eastern stream—the Aryan Hindoos—the new comers into the north of India several thousand years B.C., prided themselves on their fair complexions. Their earliest poets praised (in the Rig Veda) their bright gods who subjected the black-skinned (meaning the aborigines of India) to the Aryan white. But the influence of hot climates impresses certain peculiarities on the people inhabiting them. Thus at the present time there is a great similarity of temperament among all the races of India. It is a notable fact, that the inhabitants of Hindostan, whether aboriginal, as Bheels, Colis, Meenas, &c., or unmixed Hindoos, or descendants of Arab or Mahomedan conquerors, do not evidence those variations of temperament and constitution characterising Europeans, and especially the Anglo-Saxon race. There is the dark complexion and skin, the dark hair and the spare habit, associated with a quick intellect and irritable disposition, alternating with indolence and apathy, but yet with great powers of endurance. The system is alternately easily excited or depressed, while the functions of the skin and liver are peculiarly active, and the digestion often sluggish. Whatever may have been the characteristics of the ancient Aryans, we now find one type and one temperament more or less pervading the whole of the inhabitants of Hindostan—a temperament which may be regarded as a mixture of the nervous and bilious. There may, of course, be variations, especially in northern India; but there is rarely found, for instance, the difference which

exists between the typical nervous, excitable Frenchman and the dull, heavy, phlegmatic Dutchman.

Now theoretically it would seem, and practically it is, I think, found, that the European who in type and temperament most closely resembles the condition to which climate and circumstances (the latter comprising the survival of the fittest) have converted the native of India, would be best fitted to encounter the anæmic influences of a tropical life. And this is the European of the bilious temperament, marked by dark features, spare build, and predominance of bone and muscle, associated with the greatest endurance and the least sensibility to external impressions, and to morbid disturbances. The name (bilious) suggests a morbid habit or tendency of the liver. But the characteristics of this temperament are usually possessed without any tendency whatever to hepatic malady. The term bilious is a misnomer, and has arisen from the European of this temperament participating to some degree in the cutaneous excretion of carbon which marks the coloured races, and so assuming a more or less dark, or so-called *bilious* aspect; an appearance, however, which is quite different from that consequent on liver derangements. Sir J. Fayrer says, 'Vigorous, healthy persons of moderately spare frame, with sound viscera and temperate habits, can withstand a great amount of heat,' and persons answering to the above description are usually of the bilio-nervous temperament; but there must be *no* predominance of the nervous type, otherwise the daily and even hourly minor ills and irritations inseparable from life in the tropics will prove too distressing for the maintenance of health.

The temperament which seems next best suited for the tropics is the *sanguine* type. This is to be explained by the high animal courage and tendency to look on the best side of matters which characterises this variety. But the sanguine are far more apt to suffer from head affections, and

their great susceptibility to external impressions renders persons of this habit and disposition less qualified to cope for an indefinite period with the daily annoyances of tropical life than others of less susceptibility. The sanguine temperament, indeed, endures only for a period, and may rapidly or suddenly give way.

The remaining type — viz., the *lymphatic* temperament—marked by an inferior power of resisting disease, by tendency to disorders of the glandular system, to struma, to maladies of the liver and digestive organs, must be regarded as unfitted for the tropics. The same observation applies to the *phlegmatic*, meaning thereby an extreme type of the bilious; to the *choleric*, or extreme type of the sanguine; and to the *melancholic*, or extreme of the nervous. It is not asserted that the question of temperament is of sufficient importance to regulate the choice, for example, of soldiers for tropical service. But I consider the question of temperament should be taken into more prominent consideration than is now the practice when deciding on the eligibility of individuals for tropical life. If doubt exists in any particular case, temperament should decide, for temperament either favours or retards the approach of anæmia.

As regards conditions of life, over-crowding or absence of fresh air must be again remarked upon as a special accessory cause of anæmia. This is especially potent against soldiers sleeping many together in barracks, the atmosphere of which, notwithstanding every endeavour towards ventilation, does become vitiated, as a visit to any barrack before the men rise will abundantly demonstrate. The same cause is in operation among prisoners in jail, and among the lower classes of natives generally, who habitually sleep in badly ventilated apartments. Again, all occupations which entail a diminished supply of fresh air, great fatigue, exposure to vicissitudes of temperature, and indifferent food, also predispose to anæmia.

As regards age, anæmia is more prevalent at certain periods, especially youth and in advanced life. At both these epochs the blood is comparatively more watery and contains less solid matter, while in youth there are great demands for nutrition and growth. The desirability of growth and nutrition being matured before a person proceeds to the tropics appears to be fully recognised, although practically it is not acted upon; for sec. 18, par. 14, of the Queen's regulations for the army lays down that no man shall be sent to India before he has attained the age of twenty. The Army Sanitary Commission recommended twenty-five as the limit, and this recommendation has been reiterated by Deputy Surg.-Gen. Hewlett in a recent report on typhoid fever affecting young soldiers. In old age, again, there is impaired digestion resulting in inefficient nutrition, or, in other words, 'the blood-springs of old age dry up.' The generally anæmic condition of the children of Europeans brought up in India, so different from the robustness of the English boy and girl, forcibly demonstrate the truth of the above, and so would the condition of elderly Europeans in India were they not so exceptional, Government limiting the period of service of Europeans in the East to fifty-five years of age for executive work, and to sixty for administrative employment. And although some men are as young both physically and mentally at sixty as others at fifty, it is nevertheless the rule that in every climate between these periods the individual begins to show signs of loss of power in diminished sensibility, muscular weakness, and impaired memory, while such maladies as calculous affections, osseous deposits, or organic visceral disease are more likely to develop; and in the tropics such developments are still more prone to occur than in temperate climates. The effect of a hot climate in inducing premature ageing is not only exemplified in the European, but also in the native, who at fifty or sixty is usually an old

man in comparison with men of a similar age in Europe (*vide* p. 13).

As regards sex, the tendency to anæmia is greatest in the female, and for this there are several sufficient reasons. In the female the total volume of blood bears a smaller average proportion to the weight of the body than in the male. According to Becquerel and Rodier, 1,000 parts of blood contain on an average 141 of red particles in men and 127 in women, while the amount of albumen and serum in the male is estimated at 69·4 and in the female at 70·5. Valentin states 10·9 as the normal rate of red corpuscles found in men and women. Such variations render a transgression of the limits which divide health from degeneration in the direction of anæmia more likely in the female than in the male. Then there is the direct relaxing effect of heat on the tissues, which, from some unexplainable reason, is more marked in the female (*vide Uterine Disorders*). It must also be recollected there are in the female system additional and important organs especially subject to tropical influences. In pregnancy, again, there are alterations in the blood tending to anæmia. The blood of the pregnant woman contains relatively a larger proportion of water, with diminished albumen and red corpuscles. The drain on the system produced by prolonged lactation has already been noticed, and this leads to similar deterioration of the blood. There are, therefore, additional and cogent reasons why women in the tropics should, as they so often do, break down into anæmia before men.

Again, something must be attributed to *hereditary influence*, or to the operation of outward influences during fœtal life, infants being sometimes born anæmic. It appears certain that a predisposition to anæmia, like a predisposition to the hæmorrhagic diathesis, is often hereditary, probably developed and intensified into a morbid state by climatic influences, of which prolonged heat is the principal. It has

also been noted that individuals born of parents who have suffered from ague and anæmia are more likely to become the subjects of anæmia than others having robust progenitors.

Symptoms.—Anæmia, as generally regarded, may be acute or pernicious, sub-acute or chronic, which, however, is only another way of stating that it may be more or less rapid in its development. The most acute form of anæmia is typified by the condition which follows sudden—as, for example, *post-partum*—hæmorrhage, or by the algide stage of cholera. But the rapidity with which *pernicious* anæmia is sometimes developed from other causes is almost as great. Thus, in twenty-four hours, according to Kelsch, a patient with intermittent fever lost from the blood a million globules per millimetre cube.

The symptoms of ordinary anæmia are as follows. The skin becomes pale, and may assume a sallow appearance. In the native and half-caste (East Indian) the skin loses its brilliancy and softness and becomes of a lighter tinge, looking more semi-transparent, while the ordinarily lighter-coloured palms of the hands become much more white. The countenance is very expressive, for, in addition to the general pallor, the eyes are often encircled by a more or less dark areola, the conjunctivæ look pearly, and the lining membranes of the eyes, the nose, and the lips, instead of being rosy, are of a pale pink tint. This sallowness of face, resulting from a deficiency of red globules, is readily distinguished from that which arises from biliary derangements by the circumstance that the conjunctivæ are bluish-white or pearly, while in the latter affections the eyes are more decidedly yellow than the skin. In addition to this general exsanguine expression the countenance sometimes appears bloated or 'puffy,' while the body loses weight. But in some instances there may be predisposition to the accumulation of fat, especially about the heart; for, as

Brunton has shown, when the power of the blood to convey oxygen is lessened by the diminution of its red particles, the consequent want of oxidation leads to the accumulation of fat. The tongue is pale and tremulous, and the interior of the mouth partakes in the prevailing exterior loss of colour. The patient is habitually chilly, languid, and indisposed to exertion, and the extremities, especially the feet, are habitually cold. The urine is rarely diminished in quantity, and may in some cases be increased: it is pale from diminution of pigment, and neutral from lessening of free acidity. There is often spermatorrhœa, and in the end torpor or extinction of sexual appetite. Menstruation becomes irregular, scanty, thin, and watery, ending probably in menorrhagia. Leucorrhæa is in females an almost certain complication. As the malady progresses there is shortness of breath, especially on exertion, probably palpitations, cardiac, arterial, or venous murmurs, often *tinnitus aurium*, spots or sparks before the eyes, and heavy sleep. The previous languor and disinclination to exertion now gives place to a feeling of thorough weariness, many giving themselves up to a *far-niente* state, and this in its turn tends to weaken them still more. The appetite, at first fairly good, is now variable and perhaps depraved, while digestion becomes more and more impaired, with sometimes pain referred to the epigastrium, and sometimes vomiting after food. There may also be attacks of recurring epistaxis. The ill-nourished brain presents various evidences of weakness: impressions too feeble to be perceived by healthy persons acting with force on the anæmic. There is also much capriciousness and irritability of temper. There is loss of memory and of the power of fixing the attention, so that mental work is only performed by a painful effort. The man becomes hypochondriacal, the woman hysterical; there is great indifference to the future, and occasionally attempts at self-destruction. Other occasional symptoms are aching of the limbs, coming

on suddenly and lasting a variable time, often after a little exertion, neuralgic headaches, eczematous eruptions, and retinal ecchymoses. When the disease has existed some time, œdema of the feet and ankles may be expected, and ascites sometimes occurs.

It is not, however, to be understood that all the symptoms enumerated appear immediately, for the process of degeneration may be one of months or years. Neither do the symptoms always occur in the marked manner described, a minor degree of degeneration causing a minor manifestation in every shade and variety. A lesser degree of the symptoms described is not incompatible with apparently fair health and with the pursuit of ordinary occupations; and it very often happens that before the symptoms attain to the intensity described in the text, other changes occur, notably in the spleen, leading to the addition of *leucocythæmia* (p. 36) to the anæmic state. But the anæmic condition from heat, aided perhaps by moisture, and comparatively chilling cold at night, by sleepless nights from heat, by immoderate perspiration, by sudden chills, and by occasional extraordinary fatigue and exposure—the anæmic condition from these climatic causes is, I believe, the *first* step towards splenic deterioration of the blood. It is not, however, denied that spleen malady (leucocythæmia) may and does arise independently of heat or climatic anæmia, but it arises as a special form of malady, although giving rise to anæmia. In other words, although anæmia may exist without leucocythæmia, the latter is always associated with the former as cause and effect.

Several of the *prominent symptoms* of anæmia are now mentioned at more length with reference to their causes. *First*, as regards the peculiar pallor. Certain alterations which take place in the blood have been referred to (p. 16), but two deviations from a healthy standard appear to co-operate in producing the paleness of anæmia. The red discs

as ascertained by direct enumeration are diminished in number, and being paler in colour it is inferred they contain less hæmoglobin. A similar conclusion has been arrived at by analysis, and by Vierordt's colour-scale. It is, however, arbitrary to say what diminution of red particles constitutes anæmia. Keyes counted the red discs in six healthy adults, and in twenty-six syphilitic adults five hundred times, and concluded that 5,000,000 red particles per millimetre cube is the full healthy average, and that the diminution in syphilitic anæmia rarely passes below 3,000,000 per cube.

As with the anæmic pallor, the other constitutional symptoms presenting may all be traced to the alterations in the blood, or to the consequent interference with nutrition. Thus the cold extremities of the earlier stages, and the œdema and dropsical effusions of the latter, are attributable —the first to feeble circulation, the last to hypostatic congestion caused by a still further slowing of the blood-current consequent on increasing debility. The general chilliness is attributable to impairment of heat-generating power from the smaller quantity of oxygen carried to the lungs by the diminished number of red corpuscles. The habitual though moderate increase in the number of respirations marking the less severe forms of the disease, and of which the patient may scarcely be conscious until he undertakes some more than ordinarily violent exertion (as, for instance, ascending a long flight of steps), when he is suddenly seized with shortness of breath, and the confirmed dyspnœa of the more acute stages, are doubtless due to imperfect carbonisation of blood in the lungs from the diminished number of oxygen carriers. The irritability, muscular weakness, and loss of flesh, are dependent on impaired nutrition from poverty of blood. The occasional deposits of fat have already been referred to diminution of oxidation (p. 28). Again, the feeble circulation evidenced by the diminished force of the cardiac beats, and by a small contracted pulse, is all evidence of blood-

deterioration, and so, indeed, are the palpitations and cardiac, arterial, and venous murmurs to which the anæmic are subject.

When anæmia has lasted some time there is gradual atrophy and fatty degeneration of muscle, in which that of the heart participates. Then the sounds of the heart become more or less muffled, the anæmic murmur may be heard over the heart, and the *bruit de diable* in the veins of the neck, or as some believe in the carotid arteries. The anæmic murmurs are of three kinds—cardiac, arterial, and venous. The cardiac murmur is a bellows sound, accompanying the first ventricular sound, which it more or less masks. The sound is short and soft, and heard towards the base of the heart rather than the apex, as an organic disease murmur would be. Its maximum intensity is in the fourth intercostal space near the sternum, and its site is probably the aortic orifice, but thought by Hughes to be at the orifice of the pulmonary artery, and by Parrot in the right auriculo-ventricular opening. The arterial anæmic murmur, which is comparatively seldom heard, consists of a blowing sound, synchronous with the pulse which in the large arteries gives a thrill to the fingers. It occurs in the subclavian, carotid, femoral, and abdominal aorta, and has been thought by Virchow to be associated with a narrowing of the arteries. The venous murmurs consist of one continuous musical, humming, singing or buzzing sound, not intermittent like the arterial murmur. They are heard most frequently at the right side of the neck, at the junction of the external and internal jugulars. They may also be heard in the femoral veins. These venous murmurs are seldom absent in well-marked anæmia. None of these anæmic murmurs are of the snoring, whistling character of organic murmurs, and they are supposed to be due to irregular vibration of valves.

Pernicious Anœmia, termed also *lethal*, *malignant*, and *febrile* anæmia, should be regarded as an anæmia in

which the symptoms are more intense, a fatal termination more probable, and in which the pathology is even more obscure. It is, however, probable that anæmia, associated with other maladies, has been described as a special form of disease under the distinction *pernicious*, or *lethal* anæmia. The anæmic may be seized by sudden diarrhœa, by cholera, by remittent fever, the prominent symptoms being the sudden and marked increase of anæmia, leading to obscurity of the superadded malady. In certain examples of excessive splenic hyperæmia accompanying intermittent, the declaration of increased anæmia has been so sudden and intense as to entitle it to the term pernicious. Again, extreme anæmia may be found suddenly developed when to simple anæmia there has been added the condition known as leucocythæmia. Or when simple anæmia has been associated with the hæmorrhagic diathesis, or with the scorbutic condition of blood,—or with the syphilitic taint, or (in former days) with the spoliative system of bleeding and mercury. I confess that I am not familiar with that type of pernicious anæmia which has been described, 'for which no adequate cause can be discovered either in the patient's circumstances or in the previous state of constitution, when the usual extrinsic and intrinsic causes of anæmia are absent, when a careful examination of the kidneys, liver, spleen, and lymphatic glands shows their condition inadequate to explain the progress of the disease, where even sometimes no disproportion in the number of red corpuscles and leucocytes has been found.' On the contrary, the intense forms of anæmia with which I am familiar have afforded an exaggeration of the apparent causes and changes met with in the less intense varieties of the malady. Some other debilitating cause has been superadded, and the blood, during life, has contained not only white cells, but also many ill-shaped red corpuscles, and granules—probably shrivelled corpuscles—indicating excessive destruction of these vitally necessary agents.

In pernicious anæmia superadded to an intensity of all the symptoms of simple anæmia, there is generally great gastric disturbance, marked by hiccup, retching, vomiting, and deep-seated epigastric pain. The blood-murmurs previously described are usually very marked. Hæmorrhages may occur from the nose or bowels, and in some cases remarkable nervous symptoms supervene, which, as Marshall Hall long since pointed out, are due, especially in children, to anæmia of the brain. Thus the pupils may be unequal, defects of vision may occur, possibly due to retinal hæmorrhage, sparks and halos of light may appear before the eyes, general epileptiform convulsions may occur, and coma may ensue. Towards the termination of these cases of intense anæmia, which may last days or weeks (during which the patient lies prone from sheer debility), there is usually irregular pyrexia, sometimes purpuric spots, and before death profuse perspirations and diarrhœa. Lastly, this intense form of anæmia has been described by Lebert and others as especially attacking pregnant women. It has been already observed (p. 27) that the blood of pregnant women is predisposed to anæmia, but in my experience I have not found them especially liable to the pernicious or rapid form of anæmia. Women who over-nurse are, I believe, more liable than pregnant females.

Post-mortem appearances.— The appearances after death in genuine cases of anæmia are strictly consequences of the condition of the blood. There is the same pale exsanguine condition of internal organs which characterises the skin, gums, and conjunctivæ during life. The heart and great vessels contain very little blood, and the clots are pale in colour. Some amount of œdema is always noticed, and there may be general anasarca. There is also watery infiltration into the lungs and into serous cavities. The body is sometimes found wasted, but more often there is local persistence of fat, and the *appendices epiploicæ* are especially

noted as well-preserved. The yellow tint of the adipose tissue is also remarkable. Fatty degeneration of the diaphragm and other muscles has been noted; and usually the voluntary muscles contrast strongly with other parts by presenting a dark colour. Dr. Pye Smith[1] regards fatty degeneration of the heart in the form of zigzag lines as the most constant anatomical condition; a condition first described by Dr. Wilks in 1857, and which affects the ventricles more than the auricles. A grey staining has also been noticed in the peritoneum, pancreas, spleen, liver, or kidneys, thought to be due to sulphide of iron; as it is believed from analyses by Quincke and others, that with the diminution of hæmoglobin in the blood, the amount of iron in the tissues may be increased. Or, as is perhaps more probable, the dark sulphide of iron stain (if such it be) may be derived from preparations of steel given as medicine.

Influence on, and connection with other maladies.—Anæmia plays a most important part in the causation of many other maladies. It usually induces dyspepsia. It favours the formation of clots in arteries and veins, with all the serious consequences which may ensue. It excites hæmorrhagic tendencies of all kinds, among which purpura, epistaxis, and lymphorrhœa may be prominently noticed. Anæmia also paves the way for the scorbutic taint; it aggravates syphilis, and it often culminates in diarrhœa and dysentery. There is also reason to believe that exophthalmic goitre, if not simple goitre, may originate in the anæmic condition. It is, moreover, certain that anæmia is not without influence in many cases of insanity, especially hysterical insanity, as well as in the asthenic form which occurs during lactation, or when puerperal mania results after great losses of blood. It would seem also that conditions of anæmia specially aggravate the tendencies of the nervous system to disease. Hence the neuralgias and nervous headaches from

[1] *Guy's Hospital Reports*, 1883.

which the anæmic suffer. Lastly, it is possible that the epidemic hæmoptysis described by Manson as prevailing in China, and associated by that author with a parasite or fluke (*Distoma Ringeri*) is connected with, if not caused by anæmia, for the anæmic, particularly natives, are especially prone to pneumonia.

One of the most important consequences of anæmia is the tendency of the general malnutritions to result in *fatty degeneration and dilatation* of the heart. A heart the subject of fatty degeneration is increased in size, altered in shape, and impaired in strength, leading to regurgitation of blood with all its consequences, the effect being eventually almost the same as from obstruction at the orifices, viz., visceral congestions, especially of the liver. Diminished force of impulse is the first most prominent physical sign, the sounds of the heart being weakened in correspondence with the weakness of the impulse, and afterwards as enlargement takes place there is increase of cardiac dulness. The loss of impulsive power of the organ leads to imperfect filling of the arterial system, and consequently to visceral anæmia and further malnutrition.

Again, the anæmic condition, with its accompanying desire for repose and sleep, undoubtedly contributes in some degree not only to the formation of *boils*, but also to the more formidable malady, *abscess of the liver*. Amyloid, albuminoid, lardaceous, or waxy liver is also often met with as a sequel of long-standing cachexia. The symptoms are painless enlargement, malaise and sallowness, and urine loaded with lithates. Chyluria (p. 139) is also most frequently seen in the anæmic.

But perhaps the most certain result of anæmia is the condition known as *Leucocythæmia*, or white cell blood, which may be defined as a disease *sui generis*, in which the number of white corpuscles in the blood is greatly increased with diminution of the red. The distinction, therefore, be-

tween anæmia and leucocythæmia consists in the presence of the pale corpuscles, and it is believed that simple anæmia will progress into leucocythæmia as a further demonstration of blood-deterioration, although it is not denied that leucocythæmia may arise independently of anæmia, which, however, it quickly causes. It is generally taught that the blood-changes of anæmia and leucocythæmia are caused by affections of the spleen. And this may be so, but I am quite sure that the blood-changes of tropical anæmia more often cause the affections of the spleen than they are caused by such affections.

The white cells, characteristic of leucocythæmia, when first observed by Cragie, of Edinburgh, in 1845, were thought to be pus-globules, which they somewhat resemble; but Virchow, about the same time, described a condition of the blood due to the development of white cells as associated with splenic and glandular affections; and Bennett, immediately afterwards, proposed the term *leucocythæmia*—'white-cell blood'—which has been generally adopted. Bennett also associated the condition with an overgrowth of *adenoid* tissue in various organs, especially in the spleen, lymphatic glands, and medulla of bone. This adenoid tissue was described as consisting of lymphoid corpuscles embedded in the meshes of a retiform stroma. It has indeed been supposed that the glands named as generally connected with this disease become disintegrated, more or less of their elementary texture finding the way into the circulation. A slight amount of white corpuscles in the blood has been called *leucocytosis*. The term *splenæmia* has been applied when evident spleen disease has been associated with white cell blood; and the terms *leuchæmia* and *lymphæmia* have been applied when the lymphatic glands are affected. It is further stated by Virchow that when the spleen has been affected other constituents, foreign to normal blood. have been found in that fluid, such as lactic

acid, prussic acid, and *hypoxanthin*; a material which, according to Scherer, exists in the pulp of a healthy spleen. When the glands have been affected the blood has presented numerous small whitish granules, characterised by innumerable round granulated nuclei, generally provided with nucleoli. Such appearances, however, are difficult to detect, requiring the aid of the most accomplished chemist and microscopist. But the ordinary white cells are not difficult to detect. For with a power of 250 diameters, colourless corpuscles may be seen to form from a fourth to one-half of the number of red. Also leucocythemic blood in a test-tube, especially if deprived of fibrine, will present a greyish-white appearance on the surface, which, under the microscope, is seen to be composed of white corpuscles. Not being dissolved by ether, the suspicion of fat-globules is negatived. The separated serum is clear, and not turbid as it is from a fatty condition of blood. When the change is extreme, coagulation is imperfect, and there is more water and less solid matter. It is, however, arbitrary to say what changes constitute leucocythæmia. White cell blood is a symptom comparable with albuminuria, and white cells in the blood, like albumen in the urine, may present in every shade and degree. It has been proposed to regard leucocythæmia as established when there is one white cell to twenty red. Normal blood contains one white to about 350 red particles. In leucocythæmia the proportion has sunk to 1·6.

As before referred to, leucocythæmia has been divided into varieties according to the seat of the supposed primary or chief adenoid overgrowth with which it is associated. Thus we have *splenic* or *lienal*, *lymphatic*, *intestinal*, *myelogenic*, *spleno-lymphatic*, and even *amygdalean* varieties, the very fact of such varieties serving to show that white cell blood is not the consequence of spleen disease alone, which organ indeed may appear quite healthy. It must be held in mind that whether there is or is not

leucocythæmia, there is always anæmia when there is the former. On the other hand, extreme anæmia may be seen without leucocythæmia. The *causes* which bring about leucocythæmia are certainly not well recognised, and it is often attributed to exposure to cold or wet, the predisposition, or perhaps unrecognised *leucocytosis* lingering long until something excites it, as child-bearing or lactation for instance. I cannot, however, avoid thinking that the first step in the morbid process is deterioration of the blood which deterioration causes those adenoid deposits which have been regarded as the cause rather than the effect of the disease.

I am also inclined to regard the condition known as *Hodgkin's disease*, or *lymphadenoma*, as the same malady. For, while the same debility exists, it is also characterised by a white deposit in the spleen and other glands, leading to glandular enlargement and diminution of red particles, and, although in Hodgkin's disease it is stated that the deposit is that of fibroid indurations, still we are told that the corpuscular development of the growth takes the form of the adenoid tissue of His. (Aitkin.) And although it is stated that a great excess of white corpuscles is not met with in Hodgkin's disease, this is scarcely better evidence against the unity of the two maladies. For, as already mentioned, there is only an arbitrary boundary between anæmia, leucocytosis, and leucocythæmia; and so I think there is only an arbitrary distinction between the two deposits as yet displayed by microscopical or other experts. They both belong to the *diathèse lymphogene* of Jaccound. They might both be termed *lymphonia* or *lymphadenoid*, terms which would include all formations which are not strictly tumours, but rather *hyperplasiæ* of the tissue proper to lymphatic glands. The distinctions which have been drawn are rather of clinical than pathological origin, and cases of Hodgkin's disease with excess of white corpuscles have been called *lymphatic leucocythæmia*. Regarding Hodgkin's disease as *sui generis*,

it does not appear very prevalent in India, Chevers stating he only saw two such cases. I believe, however, I have often seen Hodgkin's disease in Indians, and been content to regard it as leucocythæmia with lymphatic enlargement.

The *symptoms of leucocythæmia* are, in addition to the greater pallor consequent on the number of white cells, a more aggravated condition of debility. The peculiar pallor is more marked, the debility more profound, and cardiac and venous murmurs are more readily recognised. The tendency to passive hæmorrhages is more marked, the spleen, liver, and other glands become enlarged, the body becomes emaciated, and ascites and œdema of the extremities more frequently present. In some cases also the gums become spongy and inclined to bleed, which many regard as caused by the disease, but which I attribute to a superadded scorbutic taint. Lastly, it may be remarked that women seem especially liable to white cell blood about the climacteric decade, and during pregnancy and lactation, as they do to simple anæmia.

Some writers think that *Addison's disease* or *melasma* may be mistaken for anæmia. The most important features of Addison's disease are progressive feebleness without any apparent cause, and a peculiar change in the appearance of the skin, which is spoken of as 'bronzed.' This bronzing may also be present on the mucous membrane of the mouth. There is usually considerable gastro-intestinal disturbance, and the patient is certainly anæmic. This malady was at first supposed to be connected with diseases of the supra-renal capsules, consisting of the deposition of a soft homogeneous substance which subsequently may degenerate into abscess. But Wilks long since showed that the supra-renal capsules may be diseased without any such bronzing of the skin as Addison connected with the condition, and a similar bronzing may occur in connection with *diarrhœa alba* (p. 168). It seems probable that Addison's disease is simply

a form of anæmia in which slight deposition of pigment takes place in the rete mucosum.

The condition known as *chlorosis* simulates to a very considerable extent the pure anæmic state, and when chlorosis occurs in anæmic girls it is impossible to say how much of the debility presenting is due to chlorosis and how much to anæmia; yet as medical practice in the two affections should be different, the diagnosis is important. The following are the principal distinctive features :—Anæmia is caused by a variety of circumstances, the principal being tropical heat, which impoverish the blood; chlorosis is induced by obscure causes generally connected with the uterine functions, and may present independently of any cause of anæmia. In anæmia the alterations in the blood are constant, and affect the whole of the constituents of that fluid; in chlorosis proper the change appears to be limited to the red particles. In both maladies there is a diminution of number of red discs, and also of colouring matter. In both diseases, therefore, the physical signs may be much alike, but it is stated by Becquerel that in anæmia the morbid murmurs are more often in the arteries (which I question), and in chlorosis in the veins. In anæmia there is constant relation between the intensity of the symptoms and the poverty of the blood, which is not always the case in chlorosis. The duration of anæmia may be regarded as depending on the causes which produce it, but chlorosis is very variable in its duration, and there is no such evident connection observed. Anæmia occurs at all ages and in both sexes, while chlorosis is limited to the young and to the female sex. While many of the ordinary symptoms of anæmia and chlorosis are nearly identical, there are some more especially characteristic of the latter. Such symptoms are the yellow greenish colour of the skin, which is different from the pallor of anæmia; the more marked halo round the orbits; the more frequent exhibition of perverted or depraved appetite, causing such

substances as lime, chalk, slate pencil, dry rice, to be sometimes greedily eaten; the more frequent complaint of throbbing pain at the top of the head; of pain in the left side, over the false ribs, not increased by respiration; of paler and more copious urine of low specific gravity, and of hysterical symptoms. Further, chlorosis is usually attended with marked pain in the back and loins at the menstrual periods. Also chlorosis does not run on to splenic anæmia or leucocythæmia, and there is no enlargement of the spleen or glands, neither does it terminate in pernicious anæmia, or give rise to the serious ailments mentioned as the sequelæ or results of anæmia.

The *debility from incipient phthisis*, which, contrary to the general view, is a common disease among the natives of India, may be confused with the debility of anæmia. But it is believed that the blood-changes of anæmia are antagonistic to tubercle. As Pollock has pointed out, anæmia wastes those organs and tissues dependent on the supply of pure red blood, while phthisis wastes all organs by furnishing them with impure blood. The phenomena of anæmia are caused by insufficiency of blood; those of phthisis by impurity of blood. Yet it is very common for anæmic patients, especially females, to be thought consumptive by their friends, and sometimes the diagnosis is not altogether free from difficulty, especially when there may be a phthisical family taint. The principal diagnostic features are the absence or presence of stethoscopic sounds characteristic of lung disease, the general appearance and history of the patient, and the loss of flesh, which is more decided in the earlier stage of phthisis than in anæmia.

The *prognosis* of anæmia is satisfactory in the earlier stages, the reverse in the latter. In many cases recovery is incomplete, and the vitiated condition of blood is never perfectly remedied. And this, apart from complications and sequelæ, which render recovery still more doubtful.

Treatment.—The most effective, and therefore the proper treatment, may be summed up in three words: *change of climate.* In comparison with radical change of climate all other means are futile. The native may be sent into districts where the rainfall is moderate and the variations of temperature comparatively inconsiderable. In the Bombay Presidency the Deccan tableland appears to be best suited for such cases. For some natives, especially Parsees, and those of the north of India, the intra-tropical hill stations are preferable. But as regards natives it is a well-known fact that many frequently become anæmic when removed from their native districts even into a more healthy locality. Their condition has been described under the term *cachexia loci*, but it arises to a very great extent from mental depression, which has already been mentioned as one of the causes of anæmia (p. 17). This mental depression, from which natives, especially Hindoos, so frequently suffer, may perhaps be better defined under the term *nostalgia*: and for natives thus suffering return to their native locality and to their homes is the principal means of cure.

When Europeans are the subjects, change of climate is a still more urgent necessity. In less serious cases certain hill stations during hot months, and the sea coast in the cold weather, may be productive of benefit. But in all probability such amelioration will only be temporary, and return to the former locality will be followed by increase of disease. Some hill stations, moreover, are not adapted to such cases. Mount Aboo, for instance, is, during the autumn season, 'malarious,' or at least the climatic conditions are those rendering persons liable to paroxysmal affections likely to suffer from recurrence. The same is true, to a lesser extent, as regards Nyee Tal and other Himalayan stations, but less so with respect to the Neilgerries. In all cases of confirmed anæmia a more radical change of climate is demanded for the European than that afforded by Indian hill ranges.

When the individual does not dislike a sea-life and is not troubled by sea-sickness, a voyage round the Cape, as taken in former days, would be usually beneficial. When, however, sea-sickness is induced by the least motion, as it is in many individuals, a sea-voyage will not be advisable. The sickness not only prevents a due amount of nourishing food being taken, but the act of vomiting has a directly irritating effect on any enlarged abdominal organ, and both spleen and liver may be implicated in confirmed anæmia. A debilitated person on board ship, even if not actually sea-sick but simply nauseated, or dreading sickness, makes little progress towards recovery. Sailing within the tropics, as frequently recommended, is productive of very little permanent benefit. For all Europeans suffering from confirmed anæmia, the change to the temperate parts of Europe should be insisted upon. But the journey should not be undertaken at unseasonable periods of the year, and the vicissitudes of English winter and spring weather should be avoided. Great attention to clothing is also necessary, and a person so situated can scarcely dress too warmly or take too great care to avoid chills. Removal to a cold climate will in many cases, if not too long deferred, result in recovery without other medicines than occasional Hunyadi Janos or other mineral aperient water. But when the removal has been long delayed, months or even years may elapse before the recovery of a healthy colour and a robust habit.

It should be noted that chronic diarrhœa marked by light stools is not unfrequently a sequel of confirmed anæmia, when the person has been incautiously removed to a temperate climate, or to the Himalayan mountain stations, and this is especially the case when the liver is also implicated. Imprudence in diet and exposure appear frequently to excite this *diarrhœa alba*, which often assumes a very serious form in the cachectic European returned from India (*vide* p. 168).

With regard to drugs, mercury in any shape or form should seldom be used. Although it has been stated that mercury at first increases the number of red corpuscles, there is no doubt that the not very remote effect is to impoverish the blood and to decrease the number of red particles, 'blanching the cheek of the rose to the whiteness of the lily.' Still, when there is evidently sluggish action of the liver an occasional mercurial aperient, by stimulating the duodenum and by reflex action on the gall-bladder, may be productive of benefit by temporarily improving digestion, and is, therefore, to be preferred to saline purgatives or to mineral waters, so often prescribed when mercurials are thought undesirable. In cases where, for any special reason, mercury is counterindicated euonymin may be used. Neither are tonics of much benefit. Iron, which is apparently required, must be distrusted; for the existing healthy corpuscles have little or no power to absorb hæmoglobin, and are, in fact, already overcharged with iron. Phosphorus has also been tried and found wanting. Probably the best tonics are quinine and arsenic. The latter has been especially lauded by Drs. Bramwell[1] and Pye Smith. But however great may be the benefit from arsenic in temperate climates, its effects are often not appreciable in tropical anæmia. More benefit is to be derived from moderate exercise every day, from tepid bathing, from free ventilation of the living, and especially of the sleeping apartments, and from good nourishing diet. Horton[2] recommends blood as an article of diet, but I think Liebig's raw meat soup much better. Whatever may be the diet it should be as easily digestible and as nutritious as possible, and with such proviso the taste of the patient may be freely consulted. It is also essentially necessary that the diet should contain a due proportion of fresh vegetable material, as scurvy is much

[1] *Ed. Med. Journ.*, 1877. [2] *Diseases of Tropical Climates.*

more liable to occur when the system is anæmic. Digestion, if failing, may perhaps be promoted by the use of pepsine with the food. As drink, good claret or burgundy or porter, or even a little good port wine, may usually be given with advantage, according to the taste or wishes of the patient.

In severe cases of pernicious anæmia both the injection of a solution of salt and transfusion have been practised, and both have been spoken of favourably and unfavourably. But in the anæmia of the tropics such operations, even if successfully performed, would only act as palliatives.

The treatment for leucocythæmia is practically that for anæmia.

Chlorosis has been described as, in some respects, in contrast, as regards its blood-lesion, to anæmia. And it is in still greater contrast as respects its response to iron treatment. In chlorosis the supply of young feebly growing but otherwise healthy coloured corpuscles is more abundant than in anæmia. It must, however, be understood that chlorosis is dependent as much on scanty absorption of iron into the system as on a deficient supply of iron; hence unless combined with well-regulated sanitary conditions and hygiene iron will do little good. But often it is very serviceable. If constipation occurs it will also be desirable to use aloetic laxatives, such as two grains of extract of aloes with a quarter to half a grain of ipecacuanha compounded with Castille soap. Then sulphate of iron will be found especially useful when the tongue is flabby, pale, broad, and indented by the teeth. In other cases neutral solution of peroxide of iron (known as liquid dialysed iron) may be used, this preparation being less disagreeable in taste than the tincture. Marriage is frequently curative in chlorosis, but the reverse in anæmia.

CHAPTER III.

BERI-BERI.

CONSIDERABLE difference of opinion prevails as to the causes and nature of this disease, and even as regards the origin and meaning of the name. Carter derives it from *bahre*, a sailor, proceeding from the Arabian word *bahr*. Others have supposed it to be derived from the Hindustanee *behri*, a sheep; the peculiar gait of this animal having been thought to resemble the movements of those suffering from beri-beri. In Japan it is known as *isiberi*, and also as *kakke*. But the word *beri* is the Singhalese term for weakness, and the repetition of the word is understood as signifying great weakness, which is the origin and meaning generally accepted.

The *pathology* of the disease is not well understood, but it may be defined as anæmia complicated with dropsical effusions. The profound and rapid anæmia which sometimes occurs has suggested to German writers (who have probably never seen the malady as it presents in the East) the question of its affinity with 'progressive pernicious anæmia.' Unfortunately beri-beri has been confused with other affections, and has been described under other names from the earliest period from which descriptions date to the latest, according as the characteristics of the various attacks more or less approached the type. Thus the older Indian authors, as Bontius and Lind, describe two diseases under the terms *barbiers* and *beri-beri*; the first a *chronic* malady, the second an *acute* malady; the one characterised by pain, numbness,

and partial *paralysis* (a very vague term) of the superior and inferior extremities; the other marked by uneasiness and partial loss of power in the extremities, œdematous intumescence, and obstructed respiration. It would appear, however, that there was no such disease as *barbiers*, for the descriptions left by Bontius, Lind, Clark, and Marshal, are either applicable to beri-beri, or to that form of rheumatic affection which has been since known as 'moon-paralysis.' Thus Bontius described the disease as 'a species of palsy in which the motions of the feet are languid and depraved.' Clark described it as a palsy with œdematous effusions, brought on by exposure to the land wind. Lind described it as a 'species of palsy, distressing those who when intoxicated sleep in the open air exposed to the land winds,' while Marshal said it commences with pain in the muscles of the thighs and legs, with numbness and imperfect power of locomotion caused by lying down in the open air, sometimes terminating in œdematous effusion. There is less matter for surprise that these older authors should have confused beri-beri with other diseases, or have described it under different names, when we know that it has since been confounded with *lathyrism*, or paralysis of the lower extremities caused by the habitual use of the seed of the *lathyrus sativus* of Central India as an article of diet; and when we learn from Barry,[1] in 1870, that his predecessors in charge of the Ceylon Rifles returned it as debility, atrophy, dropsy, rheumatism, asthma, dyspepsia, and malarious fever, according to the principal characteristics of each case; and when we find it being described at even a later period under various names, and as a 'new disease' (*vide* p. 54).

Symptoms.—In typical cases, sometimes after intermittent fever, sometimes without ever having so suffered, patients are first attacked with weakness and stiffness of the ankles, legs, and thighs, often accompanied by formication

[1] *Report on Ceylon Rifles*, A. M. R., 1870.

and more or less acute pain, worse in damp weather and usually relieved by warmth. Either preceding the attack or accompanying it, there is considerable general debility. In the course of a few days there is numbness and œdema of the legs, with tenderness in some parts, best discovered by passing a hot sponge over the surface. In the course of another few days the œdema of the extremities increases, and they feel weighty and rigid, so that locomotion is interfered with, and the person staggers and 'straddles' as he walks. Sometimes there is pain along the spine, particularly about the lumbar vertebræ. The abdomen now becomes affected, with more or less speedy extension of œdema to the whole body. Then there is great thirst and sleeplessness, weight and tension at the præcordia, palpitations, dyspnœa, cardiac bruits, and symptoms of congestion and effusion in the lungs. As the disease advances the dyspnœa increases, the face becomes swollen, the lips livid, the limbs become more œdematous, and frequent vomiting or diarrhœa, a quick small intermittent pulse, sense of suffocation, scanty and often albuminous urine, precede a fatal result. During the whole illness there is little febrile manifestation. The ordinary duration of the disease is a fortnight.

The condition of the urine varies much. It is generally clear and slightly high-coloured, becoming more so as death approaches. It is often of low specific gravity and normal acidity; more rarely alkaline, of high specific gravity, and depositing abundant phosphates. It is rarely albuminous at first, unless from some prior renal defect, although, as previously mentioned, often found so eventually.

All cases of beri-beri, however, are not of the typical nature sketched above. The œdema may be so acute as to simulate acute general dropsy; which, indeed, it is, occurring in a subject debilitated by the causes to which beri-beri is presently referred (p. 53). The œdema may rapidly spread over the body, and death may take place in a few days. Or

the malady may be chronic, advancing during weeks or even months; the first stage of weakness, numbness, or pain in the lower extremities lasting an indefinite period before the occurrence of œdema. It is these cases which are liable to be mistaken for rheumatism, or for incipient paralysis. The acute stage has been described by Simmonds (Yokohama) as *beri-beri hydrops*, or 'wet beri-beri,' and the chronic form as *beri-beri atrophia*, or 'dry beri-beri.' By Ramnay Roy the forms have been described as *inflammatory* and *asthenic*. Between these extremes there may be every conceivable degree and form of the malady. It is impossible to define any particular course the early symptoms of beri-beri may take. They may rapidly proceed to a fatal termination by general anasarca; or there may be partial recovery, the anasarca being reduced, although the difficulty of locomotion (which has been mistaken for paralysis) remains. In other cases, when the disease assumes a chronic form, the progress is slow, and the œdema may be long confined to the legs, or even to the front of the tibiæ. It is in these protracted cases that visible scorbutic symptoms are sooner or later developed.

In the œdematous form, occurring among the famine-stricken, general dropsy may be the first symptom, but in one epidemic which came under my cognisance in Gugerat, after the famine period of 1877–78, the dropsical effusion was more or less, and in many cases quite limited to the abdomen, and numbers were tapped for ascites. Other symptoms occasionally occurring are 'earth-eating' at an early period; also burning of the hands and feet. Sometimes the numbness, or burning, or pain, occur periodically. Occasionally, but not very often, dyspepsia is a prominent complication in the chronic form. Or the case may be marked by the occurrence of intermittent fever. The gums are often scorbutic, and petechial spots may present. The dyspnœa is also very variable; sometimes scarcely recognised, at others

forming the principal complaint of the patient. Epistaxis, hæmorrhage from the bowels, dysentery, or bloody urine may complicate the case. Sudden death often takes place from either failure of the heart, incommoded by pericardial effusion, or from embolism. Congestion of the lungs frequently causes a fatal issue. During convalescence there may be much desquamation of the cuticle.

Classes most subject to Beri-beri.—It would appear the natives of India are especially liable to the malady, for in addition to its being found throughout Hindustan, Indians were chiefly attacked by that form known as the 'acute œdema of the Mauritius.' Also natives of India in the Persian Gulf and Red Sea are very liable. It scarcely ever attacks persons under adult age. Ramnay Roy says out of 650 instances he observed it in only three children under seven, and in none under four. Males are more susceptible than females—a case occurring in a female not having been noticed by any of the older authors excepting Malcolmson. This may, perhaps, arise from the military surgeon's duties being so much confined to males; but that it does happen to females I know, having seen instances at Bhooj in Kutch, at Bassadore in the Persian Gulf, and especially during periods of scarcity in Rajpootana. I have never seen a European affected, but believe instances of the kind do occur. Marshal states, in 1820, many cases presented among European troops, but our military sanitation is better now than it was then, which doubtless prevents the disease. Among natives the number of attacks and the mortality are twice as high among those who are ill-fed, badly-housed, and poorly clad. Although the cachectic are generally the victims of the malady, it yet occasionally occurs in seemingly healthy men, and in many instances it has been known to prevail with epidemic violence among bodies of soldiers and sailors, the instance of the Deolee Irregular Force, referred to at p. 55, being illustrative. Its prevalence in the Carnatic in

1872, in the Allahabad jail in 1881, and in Singapore in 1877–78 are other instances. And so are, I believe, the acute dropsies afterwards referred to as prevailing some years back in various parts of India and in the Mauritius (p. 54). The epidemic prevalence of the disease has led to the idea that it might be contagious, but of this there is no evidence.

Localities where prevalent.— The older authors, as Hamilton, for instance, stated it is confined to the coasts in India, and is never seen more than fifty miles inland. Mon. Durodie, in his recent contribution to the study of the disease, has reiterated this fallacy, which doubtless originated from the interior of Hindustan not being so well known in former times as it is now. I have, however, seen it in various parts of India. A detachment of the Deolee Irregular Force stationed, in 1873, at Shoojeanghur, at the triple border of the Bickaneer, Jeypoor, and Marwar States, in the semi-desert district of Western India, suffered considerably from beri-beri,[1] and I have frequently met with isolated cases in Marwar and other parts of Rajpootana. It is also stated, and endorsed by Mon. Durodie, that a residence of six months where the disease is endemic is required to develope it. This I do not agree with, having seen it developed among sepoys in the Persian Gulf, and in others in Rajpootana in as many weeks. Still it may be admitted that the littoral in India is most favourable to the malady. Hence it is found prevailing chiefly on the Malabar coast, in the Carnatic, and in Ceylon. It also occurs in British Burmah, in Japan, in China, and according to Fayrer it is known on the West Coast of Africa as 'the sleeping sickness.' The disease appears to be unknown in Europe, the only notice among the older writers of anything resembling it being in Sir J. McGrigor's account of the 'Diseases of the Peninsular Army.' But more recently Biermer has described a pernicious form of anæmia which seems to be nearly identical.

[1] *Ind. Med. Gaz.*, Jan., 1874.

Influence of season.—This is marked, by far the greater number of cases presenting in the rainy season, and less in the hot weather, during which the temperature is more equable, vicissitudes less, and damp at a minimum. The larger number of cases occurring towards the end of the rainy season in jails was noticed by Malcolmson years back, and has since been an ordinary matter of comment, being frequently used as an argument by those regarding beri-beri as a manifestation of malaria.

Causes.—Among authors who have written on beri-beri, Rogers (who first described it scientifically as *hydrops asthmaticus*) looked upon it as a dropsical effusion commencing in the chest. Malcolmson, in his prize essay on the subject (1833), considered it as a rheumatic affection in which the spinal cord was primarily deranged, the dropsical symptoms and so-termed paralysis being secondary results. Rowell (Singapore) says it is the result of a malarial poison acting primarily on the spine, depressing the vital powers, and deteriorating the blood. Rose and Barry (A.M.D.) refer it to malaria in the worst and most concentrated form. Massey (A.M.D.) mentions it as a paralytic malarious cachexia. Scott said it seems to be one of the numerous modifications of acute dropsy, and he pointed out the resemblance in the countenance and general appearance of those affected with beri-beri to dropsical maladies after measles or scarlatina, a resemblance which has recently been noticed by Chevers. Ranking thought beri-beri a form of renal anasarca. Fayrer regards it as a condition of profound cachexia rather than a specific disease. Chevers has recently described it as a specific fever *sui generis*, under the name *febris exanthematosa orientalis.* Some, as Morehead, imagine the scorbutic diathesis to be the predisposing cause, rendering the system more prone to serous effusions on exposure to cold, particularly if the kidneys, from congestion or structural defect, cannot readily take on a compensating

action. Sir Guyer Hunter regards it as the result of malarial and scorbutic cachexia combined, looking upon the paralysis as of mechanical origin. It is indeed the loose use of the term 'paralysis' which has caused much confusion in the recognition of the different forms of the malady, and the term should not be applied to the symptoms presenting, any more than it should be applied (as has been the case) to the form of rheumatism previously mentioned (p. 48), which has been mistaken for beri-beri, and has been since known as moon-paralysis (Chap. XXIV.). For in beri-beri there is no paralysis properly so called, the difficulty of movement of the limbs being due *first* to hard effusion into the muscles, as occurs in scurvy, and *secondly* to mechanical pressure from effusion within the spinal column. The definition of beri-beri has been given (p. 47) as anæmia accompanied by dropsical effusions, and under this definition the acute œdema met with so frequently several years back in Calcutta, Allahabad, and their neighbourhoods, also in the Bombay districts need not have been described as 'acute œdema,'[1, 2] or as 'acute dropsy, the new disease,'[3] but might have been classed under the old term beri-beri, as, indeed, it was described by Assistant-Surgeon Ramnay Roy.[4] Similarly the 'acute anæmic dropsy'[5] of the Mauritius of 1878 appears to have been nothing more nor less than beri-beri. Doubtless the former is a more scientific term than the latter, and is to be preferred for at least some of the forms of beri-beri. There is no doubt, in my mind, that all the affections mentioned above were the same disease, although the different outbreaks represented different phases of the malady, the anæmia and dropsy being the essential and common features. For beri-beri, as already stated, like many other maladies, does present very different and manifold conditions.

[1] Deakin, *Ind. Med. Gaz.*, May, 1880.
[2] Chambers, *ibid.*, April, 1880.
[3] O'Brien, *ibid.*, May, 1879.
[4] Roy, *ibid.*, May, 1880.
[5] Lovell, *ibid.*, May, 1880.

Personally I believe beri-beri to be a form of scurvy, the symptoms of scurvy being marked or overpowered by superaddition of others, which, when presenting in certain forms, have been termed beri-beri. In the article on scurvy it is shown that this disease may be present in a latent form, and that affections of the gums, usually regarded as essential characteristics of scurvy, are but one of its manifestations. But in beri-beri there is very often, perhaps most frequently, some affection of the gums. Thus Ramnay Roy states, 'Scurvy exists in a certain number of cases, but not in all.' In the epidemic in the Singapore jail in 1878 scorbutic symptoms appeared in some cases. In the description of 'Acute Dropsy, the new disease,' previously referred to (p. 54), O'Brien states, 'Scurvy was associated with the dropsy in many cases.' The opinion of Morehead was that beri-beri is a general dropsy coming on in the scorbutic condition. Chevers says, 'Scorbutus should always be looked for, and regarded as a grave complication.' If, as is so often the case in India, there is a latent scorbutic condition, the application of other blood-deteriorating agencies will be likely to develope beri-beri. The case of the Deolee Irregular Force (p. 52) may be again referred to as an example. The men were marched some 150 miles from the S.W. to the N.E.; from a comparatively black fertile soil to an arid sandy waste; from good to indifferent food; from good dwellings to brushwood huts; from fair water-supply to brackish; from cantonment life to harassing picquet duty; from a place were fresh vegetables were procurable to a locality where there are few, if any; and as a consequence they, being already probably affected by latent scurvy (*vide Scurvy*), became anæmic and then dropsical. Epidemics in jails, where there is the additional factor of depression of mind, may also be accounted for by confinement, hard labour, food just sufficient to support the system on scientific principles, and perhaps overcrowding, acting

probably on systems already undermined by the prevalent, although latent, scorbutic taint. Very similar remarks apply to famine-stricken populations, on whom also various other adverse influences must act as the consequences of the conditions they are subjected to. When the malady occurs generally or epidemically there are always some general predisposing causes, the principal of which are scarcity of and probably brackish water, which, imbibed at other times with comparative impunity, exerts a more injurious effect on systems below par. When the malady occurs locally, causes such as mentioned affecting the Deolee Force will always be found to have been in operation. Thus, for the epidemic in the Thatyetmyo jail in 1881 a complete change of all surroundings and circumstances was found to be the only means of checking the disease.[1] Chevers, than whom no one's opinion is entitled to greater respect, objects to beri-beri being regarded as a form of scurvy, because it is often epidemic, which scorbutus cannot become; because there was no beri-beri among the scorbutic mariners of the last century; because in many cases of sea-scurvy treated there is no evidence of the existence of beri-beri; because, although beri-beri occurs in some localities notorious for scurvy, as Aden, it does not prevail in certain districts in which scurvy is extremely prevalent. All this may be true. But if beri-beri is, as I regard it, a phase of the development of that Protean malady scurvy, the arguments fall to the ground. There is not more difference between the symptoms of beri-beri and scurvy than between those often presented by two cases of enteric or scarlet fever.

Lastly, beri-beri has been attributed to a microbe said to be found in the tissues, and reported to resemble a microbe found in rice (De Lacerda). Whatever may be the real cause or causes, it is certain the affection most fre-

[1] *Rep. San. Com. Gov. of India,* 1881.

quently follows exposure to cold, night land-winds, damp, and atmospheric vicissitudes; and that those individuals who are in a cachectic state from scurvy, fever, from the use of unwholesome food, from want, debauchery, or long confinement in jails, or on board ship, or in crowded barracks, or from the use of impure, especially brackish, water, are most liable to attack. Now, it is under precisely similar conditions that scurvy occurs (*vide Scurvy*), and I cannot but regard the two maladies as identical. In my 'Manual of Family Medicine for India,' I described beri-beri as 'the form of scurvy known as beri-beri,' and I think the term beri-beri should be altogether expunged from the nomenclature of disease, or at least relegated to what it really is—viz., a phase of scorbutus.

Post-mortem appearances and pathology.—A remarkable degree of fat is often found in different parts of the body, even after long-continued disease, and in some cases an unnatural deposit. The blood is watery, poor, and deficient in red globules.

The cellular tissue is gorged with serous fluid, and the same is found in the cavities of the chest and abdomen, in the cranium, and in the pericardium. The kidneys are more or less congested. The liver has been sometimes found chocolate-coloured, but as usually dark and congested; as is also the spleen. Redness about the lumbar and sacral nerves has been noticed. The *pathology* is very obscure, but the dropsy may be considered of a passive nature, arising from the congested kidneys being unable to pass off the watery constituents of a deteriorated blood, an accessory cause being checked cutaneous perspiration.

Treatment.—Hamilton and Maxwell thought it congestive or inflammatory, and used calomel and bloodletting. The patient was at once bled, and put on a course of calomel and squills with salines and antimonials, while the strength was supported with cordials, generally gin-punch; but as

Christie observes, 'The method of treatment was pursued with little cause for congratulation on success.' Colquhoun, Christie, and Hamilton then considered the disease due to debility, and consequently requiring a stimulating treatment only, and this plan was in vogue when I first entered the Indian service thirty-three years ago. Ridley treated his patient with diuretics and hydragogue cathartics. Dr. Maxwell imagined beri-beri allied to cholera in its nature, and, not very logically, recommended phosphorus until the motions became luminous. It may be readily understood how the different characteristics the disease presents at different times and to various observers may have led to very different lines of treatment as above mentioned; but between cholera and beri-beri I can only recognise one point of similarity, viz., as in cholera scarcely any two cases require the same treatment, so in beri-beri each case should be duly considered, and the choice of remedies made according to the symptoms presenting. *Antiscorbutics must be always freely administered*, and a strengthening but easily digested diet allowed. Stimulants will generally be of service. As regards medicines, diaphoretics are usually advisable.' If there is no albumen, or castes, or blood in the urine, diuretics may be used, the best of which are nitrate of potash and tincture of squills. Purgatives, especially drastic cathartics, should not be used, as the attempt to remove the effusions by such means is never successful, and there is always great tendency to diarrhœa and dysentery, which purgatives aggravate. External applications do not appear to do any good; nevertheless, with the desire of impressing the patient that all is being done which can be done, I usually used mild counter-irritation over the kidneys in the shape of dry cupping, mustard leaves or iodine paint. Formerly it was the custom to deplete locally with leeches or by cupping, but I soon ceased this practice.

A preparation called *treak tarrok*, consisting of eleven different ingredients, the chief being opium, junipers, and

gentian, is a favourite remedy among the Arabs on the shores of the Persian Gulf, and also among the native doctors of India. Under its use the patient passes several watery stools daily without debility being produced. The native hukeems also use a shrub called 'paychora' which acts as a hydragogue cathartic. Malcolmson used 'ol nigrum,' made from the seeds of a plant growing in the Circars called *malkungui* (celastrus paniculatus). The dose of this was twenty drops with benzoin, cloves, and nutmegs.[1] Oleum petroleum has also been recommended.[2]

The rational treatment, however, previously recommended is more successful than so-called specific remedies. But still better than any kind of medicine are good nourishing diet, and *removal* from the locality in which the disease was contracted. It is on record that the men of the Ceylon Rifle Corps usually recovered when sent out of the island, of which the majority were not natives; and when serving in the Persian Gulf we found the best method was to send the men affected away.

[1] Waring, *Ind. An. Med. Sci.*, 'On the Indigenous Medical Plants of India.'

[2] Arokeum, *Mad. Jour. Med. Sci.*, vol. xiii.

CHAPTER IV

BOILS.

THERE are *three* classes of boils prevalent in India, the *first* occurring in new arrivals from temperate climates; the *second* presenting in older residents; and the *third* usually regarded as specific.

The first variety of boils do not differ from those ordinarily seen in Europe, and they generally occur to plethoric Europeans who on entering the tropics continue the same dietary, or even consume more carbonaceous food than they did at home. Boils were mentioned by Peet as an 'insignificant ailment,' but this is not the case, for although the description of boils referred to above may not be dangerous, they are sufficiently painful, and although they may eventually prove beneficial by cleansing the system, or even preventing liver disease, they may occur to such an extent as to weaken the system, and to initiate that anæmia or blood-deterioration from which so many Europeans suffer. The *treatment* of this class of boils consists in local applications of a soothing nature, evacuation of the matter when it points, moderate purgation, and low diet; taking care that drastic purgatives are not used if cholera prevails; and that low diet is not too much insisted upon if there is any tendency to anæmia.

The *second* variety of boils is a more important disorder, as they occur to Europeans some time in the tropics, and must be regarded as demonstrative of blood-deterioration. These boils present singly or several at one time, or in successive crops. They may be of various sizes, from that of a pea

to the bulk of an egg, or larger. Large boils most frequently present on the limbs, at the back of the neck, in the armpits, or about the buttocks, and are often sluggish and long before suppurating. Small boils most frequently present on the scalp, where sometimes hundreds may be counted. Boils often attack persons who have changed their residence from one part of India to another. For instance, after a long period spent in the dry Upper Provinces, sudden change to the moist climate of the coasts is often followed by boils. In children boils may attend the process of teething. Otherwise the cause of these Indian boils is in all instances blood-deterioration resulting from length of residence, heat, poor food, scurvy taint, impure atmosphere, over-work, or attacks of debilitating fever, of which boils may be a sequel. In persons predisposed by such influences accidental local injury will often excite them. Boils are sometimes erroneously attributed to eating mangoes, the fact being that the mango season, or shortly after the mango season, is the period of the year when, owing to the intensity of the heat and the resulting depression of vitality and blood-deterioration, boils are most common. The blood imperfectly elaborated is not freed from excrementitious matter, is in a condition ministering imperfectly to nutrition, and prone to fibrous coagulations, which, as Fayrer has pointed out, cause capillary embolisms, giving rise to local starvation, and death of minute portions of areolar tissue in or under the integument, resulting in suppuration, which is set up for the purpose of getting rid of the dead fragment or core.

The *treatment* of these boils is constitutional rather than local, and the condition of general health should be attentively considered. If the tongue is furred and the digestive organs out of order, mild aperients will be required. If there is reason to suspect scorbutic taint, or even if no signs of scurvy are visible, and the person has been in a locality where fresh vegetables are scarce, or the water brackish,

anti-scorbutic diet should be employed. If there is similar reason to suspect malarious taint, quinine. If there is a syphilitic taint, iodide of potassium will be advisable. If no particular taint is evident, iron will generally prove beneficial. To all the above liquor arsenicalis may be added with advantage. It is immaterial what local treatment is employed. Large boils will generally require poulticing, and small boils water-dressing, and all should be pricked as soon as matter forms. Spirits of camphor applied every three hours, if used sufficiently early, will sometimes disperse sluggish boils. If boils persist, removal into a cooler climate will be necessary.

The *third* class of boils have been regarded as specific, and some are erroneously known as the 'Delhi sores,' implying that they are peculiar to the city so named. But this boil or sore is not limited to Delhi, for the so-called 'Sind boil,' 'Gwalior ulcer,' 'Surat boil,' 'Burmah boil,' and the 'Moultan and Lahore sores,' are very similar, and I believe identical. A similar sore is indeed met with throughout the whole of Western India, especially in the semi-desert districts of Rajpootana, and on the Malabar coast. There is also reason to believe that the 'Aleppo Biskra,' the 'Crete bouton,' and the 'Persian ulcer' are the same, and from personal observation I feel sure the 'Baghdad boil' and 'Aden sore' are so. Quite recently it was reported boils were prevalent at Suakim—'very painful and of a type similar to the Punjaub and Sind boil.' Carter, while proposing *mycosis cutis chronica* as a name for the Delhi sore, states the latter only differ from Baghdad and Aden sores in accidental features. Fox and Farquhar, recognising this, proposed the term 'Oriental sore,' so that all might be included under one appellation, and local names be avoided.

The 'Delhi sore,' although known long before as 'Arungzebe,' from the name of a Mogul emperor who suffered therefrom, did not attract much attention in India previous

to 1858, at which period the palace and city of Delhi were occupied by European and native troops. The military cantonment, previous to the mutinies, had been situated two miles outside the city walls, and although cases were known to occur in the city, the disease was rare among the military. After 1858 the troops in garrison were admitted into hospital for the sore to the extent of 40 per cent., while numerous slight cases were treated out of hospital. The sore appears most frequently, but not invariably, on exposed parts of the body; at first as a small pimple, or like an irritated mosquito-bite, extending gradually round a hair-follicle as its centre, and perhaps remaining in the same condition for days or weeks. At length a thin fluid escapes from the elevated surface, which dries and forms a scab, while the surrounding parts feel 'boggy.' Under this scab ulcerations and suppuration take place, and when the crust falls off or is removed, an indolent ulcer is exposed, with undermined edges and lobulated or fungoid-like granulations in the centre; in healthy subjects red and florid; in the cachectic (as sufferers usually are) paler or even blue. The healing process is slow, the ulcer sometimes remaining open for a month, or even upwards of a year, or longer, when gravely complicated with leprosy, secondary syphilis, miasmatic fever or a cachectic state produced by other diseases, or by famine. There always remains a depressed cicatrix, and if on the face they may give rise to deformity. In Baghdad they usually do occur on the face, and the mark called the 'date-mark' (from the malady having been supposed to arise from eating dates) is an additional feature in the physiognomy of most of the inhabitants of that city. When the sores are very large or multiple they compromise health *per se*.

When the Delhi sore is cut into, minute yellowish points are seen, which have been described by Smith (A.M.D.) as the ova of a parasite, and by others as of vege-

table origin. Fayrer, however, regards this as an abnormal development of connective-tissue corpuscles. Fleming said there is shown under the lens small yellowish bodies in glistening capsules, consisting of a fibrous envelope, composed of concentric laminæ, the contents being fluid. Carter describes a presumed parasitic organism, consisting of *spheroids* and *mycetoma*. Nothing like the bacillus of anthrax has yet been discovered.

Bad water, soil, or food; bites or stings of insects; and animal and vegetable parasites have all been charged with producing this disorder. A commission of enquiry at Delhi, in 1865, formed the opinion that the sore is of parasitic origin, and that the foul water of the city is the *habitat* of the parasite, which finds entrance through any abrasion of the skin—as from a mosquito-bite—when the person is bathing or washing; development of the parasite, inflammation, and sore resulting. Since then Fleming [1] and Smith supposed they had discovered the germ which they regarded as animal: but as Surg.-Gen. Murray (who was President of the commission above referred to) remarked. 'As Drs. Lewis and Cunningham, after careful examination, failed to see it, implicit confidence cannot be placed in less experienced microscopists.' The last researches into the subject are Cunningham's,[2] who states, 'The essential feature in the diseased area was clearly shown to be an accumulation of lymphoid and epithelioid cells among the normal tissue-elements,' which he is inclined to regard as an organism of a mycetozoic nature, concluding that Delhi boil may be associated with or possibly caused by the presence of these peculiar parasitic bodies without their being the only cause. Others, discarding parasitic origin, have regarded it as a local manifestation of a cachectic condition, due to residence in unhealthy localities. Others have regarded it as scorbutic. I believe

[1] *Ind. Med. Gaz.*, 1869.

[2] *Scientific Memoirs by Medical Officers of the Army of India*, 1886.

it to be ordinary furuncular capillary embolism, aggravated by either scurvy or syphilis, or both, or in some instances by a lupoid or leprous condition of blood. An outbreak in the 36th Bengal N.I., similar to the Delhi sore, was certainly mainly scorbutic.[1] So was an outbreak of sloughing ulcer among the 15th Sikhs.[2] The manner in which a syphilitic taint will complicate any skin affection is patent. Lewis and Cunningham, describing lymphoid nucleated cells in the sore the product of a condition they considered identical with lupoid, suggested *Lupus endemicus* as the name for the affection. And I am sure I have seen leprosy and the sore combined in the same individual. I therefore do not believe in the presence of a special parasitic organism. From the report of the Commission previously mentioned the Delhi sore would seem to be sometimes inoculable, but I believe when this occurred it was syphilis that was so transmitted, and not the Delhi sore. According to Carter, the Aleppo *bouton* is sometimes inoculable, and probably from the same taint. Fayrer states that animals are liable to the sore, especially dogs, which contract it on the nose; and dogs are also liable to syphilis on the nose. Fayrer also regards the 'Bursattee,' or rain-sore of horses, as the same malady, which may be correct.

Treatment.—The Commission on Delhi sore recommended the destruction of the vitality of the supposed germ in the earliest possible stage, by the application of the actual cautery, or by *potassa fusa* or nitric acid in the more advanced stage, with great attention to the use of pure water. But it would appear that perfect cleanliness, the use of carbolic acid dressing, and constitutional treatment as may be required, is quite as efficacious. Powdering the ulcers with burnt alum may be tried. Cases have been reported where the ulcers were thus covered with a thick, firm,

[1] *Ind. Med. Gaz.*, 1874. [2] *Ibid.*, 1884.

dry crust, which fell off in two or three weeks, leaving a surface covered with small healthy granulations.

The constitutional treatment should be that indicated for the second variety of boils, with special reference to any constitutional defect, which will rarely be found absent. Removal from the locality to a cooler atmosphere is, however, the principal means of cure. The cure of natives who have bad sores, who cannot be well fed, and who cannot be moved to another locality, is almost hopeless, and this is constantly exemplified at Aden. The preventive measures are general sanitary arrangements, and especially the supply of good water. The improvements in such respects which have taken place in India during the last quarter of a century have much reduced the prevalence of the so-called Delhi sore, not only at Delhi but elsewhere.

CHAPTER V.

BURNING OF THE FEET.

An affection which has been so termed is noticed by different authors, and has been thought allied to *beri-beri*, or indeed, according to Hunter,[1] the first stage of that disease. Mr. Waring[2] stated it occurs occasionally as an idiopathic affection, unconnected apparently with any disease; but that most generally it appears as a sequence of fever, bowel complaint, rheumatism, or beri-beri. Malcolmson regarded it as an indication of nervous debility produced by various diseases, or anæmic states of the system. It seems, however, to be rheumatism, sometimes syphilitic, of the plantar fascia. It exists in various degrees, from an uneasy sensation to the painful extreme of burning, preventing sleep, and thus destroying the general health. As the disease advances, there may be distinct exacerbations and remissions, the pain, like that of one form of rheumatism, being worse when warmly covered at night. Emaciation and debility progress, and bowel complaint probably sets in, from which at last death takes place. Mr. Playfair[3] remarked that the patients describe their sufferings as nearly insupportable, and there is scarcely a disease in which the sufferer is so speedily exhausted. The same author distinguished two varieties: one in which the parts are in a constant state of moisture from sudor, the

[1] Hunter, *On Diseases of European Sailors, &c.*

[2] Waring, 'On Burning of the Feet,' *Mad. Quart. Jour.*, No. 2.

[3] *Mad. Quart. Jour.*, vol. i.

other in which the extremities are dry and sometimes scaly. It has been remarked as occasionally accompanied by *urticaria*, but this would appear accidental. Although usually confined to the feet, the hands also are sometimes affected, which seems additional reason for regarding it as rheumatism. Occasionally there is nervous spasmodic twitching. In some instances I have known the burning alternated with numbness, or with sensations of pins and needles, and this form of the malady most frequently occurred in females.

The disease is most met with in Arracan, Burmah, and the more easterly districts. It is also said to be prevalent in China among labourers who stand on marshy ground where rice is cultivated.[1] It is also said to be known among Asiatics in Natal. In India natives are those generally affected, but Europeans sometimes suffer, as happened to a sailor under my care when in the Persian Gulf. Mr. Waring stated the disease is often feigned by sepoys, and such malingering is said to be common among coolies, which may account for its supposed occurrence among Asiatics in Natal, and elsewhere out of the East. But, as in uncertain cases of chronic rheumatism, if the man sleeps well and his appetite remains unimpaired, if he continues in his usual condition of body without emaciation, or evident diminution of strength, then the surgeon will be justified in sending him to duty. It may, however, be well to mention that sepoys, sometimes by starving themselves, induce not only emaciation, but also a febrile condition, and in such cases it is necessary to take measures to see that food is eaten.

The *treatment* is empirical and therefore unsatisfactory. Dr. McKenna found tonics most useful, and of these arsenic most efficacious. Opium or chloral is often necessary to relieve pain and secure sleep. The remedies usually given for rheumatism may be tried. Bathing the feet in strong brine is often beneficial, but at other times mustard and

[1] *Lancet*, July 14, 1883.

water appears to suit best. After bathing in either, the feet should be rubbed dry, oil should be applied (grass oil being the best), and the parts should be wrapped up warm. A poultice of dhatura-leaves is often beneficial. The natives use as a local application a mixture of salt, oil of sesamim, and lime-juice, and they fumigate the feet with the smoke of wood and Mudar-leaves (*calotropis gigantea*). Tincture of aconite applied locally has been recommended, as also various liniments of a soothing or benumbing nature. Dry cupping, galvanism, electricity, have been tried ineffectually. A blister to the instep, and the vapour of decoction of poppies was advised by Grierson; but the accompanying cachexia generally forbids any application, such as a blister, likely to degenerate into an open sore. Of all means calculated to effect a cure, none is so speedy in its results as change of climate.

CHAPTER VI.

CHOLERA.

Native names: *Jurree-Murree* (sudden death); *Maha-Murree* (great death).

CHOLERA may be defined as a disease always present in India, and frequently becoming epidemic, generally characterised by purging and vomiting usually of a material resembling water in which rice has been boiled, commonly accompanied by cramps, and frequently resulting in suppression of urine and collapse.

The term cholera involves a principle, but that principle is repugnant to the disease. The term cholera signifies a flow of bile, but the want of bile is usually one of the most prominent peculiarities. The term cholera therefore cannot be used without pointing out the absurdity of an expression which literally implies not what it ordinarily is, but what it ordinarily is not.

Allan Webb[1] long since remarked that much of the mystery and obscurity which attaches to cholera results from its having been regarded (since 1832 in Europe, and since 1817 in India) as an entirely new and previously unheard of malady. And he shows by quotations from Sushruta, Wangshoo, and other ancient authors, that cholera is as old as India or China. 'It is not always the same in its nature or effects. It is allied in its varieties to some of the most desolating pestilences, as well as to some of the most common diseases.' In its virulent form it begins, as Majendie ob-

[1] *Pathologica Indica.*

served, 'in death.' On the other hand, as with all other diseases, there may be a most mild, and scarcely recognisable form of the malady.

Macpherson [1] has elaborately and satisfactorily shown that cholera is one of the most ancient diseases of which distinct descriptions exist, prevailing *both in Europe* and Asia from the earliest times. In support of the view of cholera having prevailed in early times in Europe, Macpherson refers to Hippocrates and Galen, who described a malady very similar to cholera, and he further shows that cholera, in one or other of its diverse forms, has been described by successive European authors almost from the time of the ancients mentioned to the present date, and especially during the 14th, 15th, 16th, and 17th centuries. That cholera has been looked upon as a new disease in Europe since 1832, may be explained by the facts that medical writings of a reliable nature referring to former times were few, and that many of the writings extant, in consequence of the extraordinary variations which occur in the manifestations of the disease itself, do not describe that typical cholera which occurred in 1832, and which has come to be erroneously accepted as the only phase of the malady. Even analogous reasoning would teach that cholera must have prevailed in Europe and England in former times; for as Surgeon-Major Waters [2] points out, considering that cholera is endemic only in countries where fevers of a malarial character are invariably to be found, and that these were common in England till a comparatively recent period, it may be inferred that when the latter prevailed in full virulent vigour, the former occurred periodically, as it does in India. In support of the view of the antiquity of the malady in the East, there is the fact that the early Sanskrit writers were apparently well acquainted with cholera. In the 'Midan' of Sushruta there is a description of a dis-

[1] *Annals of Cholera.*

[2] *Lecture on Epidemics at the Sassoon Institute, Bombay,* 1884.

ease termed *Vishuchuka* (meaning purging, vomiting, and fever) which was clearly cholera, while in other old Hindoo medical works we have *jwar antishar* (meaning fever and incessant purging). Then there is the significant fact that all the different languages of the peninsula of India, to the number of thirteen, have a time-honoured name for the disease. Further, it appears to have been well known to the Mahomedan conquerors as *haidsa* or *haiza*, and it has been shown that cholera prevailed among the Mahomedans of Delhi in 1325. There is also a record in Todd's 'Rajasthan' that temples to 'Oola Bebee,' the goddess of cholera, were found by the first European visitors in India. C. Macnamara also mentions a temple to 'Oola Bebee' as existing many years in Calcutta. Macpherson makes the pertinent observation, that as it is certain the Portuguese found cholera in India soon after their first arrival in 1500, especially at Goa in 1543, it is only reasonable to believe it existed in India previously. Mr. Gaskoin has translated from the Portuguese a distinct notice of the disease occurring in 1503, when, as testified by Gasper Corea, 20,000 men belonging to the army of Zamoryn, king of Calicut, died 'sudden-like with pain in the belly.' But the first full account given by a European physician, was by Garcia D'Orta of Goa, who in 1563 pointed out several varieties of cholera much as they exist now. The ideas, therefore, which have been held by some cannot be regarded as tenable, viz. that Bontius in 1630 was the first writer on cholera in India; that cholera is a comparatively recent disease; and that, as concluded by the Constantinople cholera congress of 1854, some change came into operation about the year 1817, giving rise to the first great Indian cholera epidemic.

Even assuming, as Bryden held, that Bontius in 1630 first described cholera, I have before me references to 15 distinct notices of the disease from that period to 1784. Dr. Waters indeed states there are no less than 60 separate and

independent testimonies to the occurrence of cholera in India during the 16th and 17th centuries. Lastly, there is no doubt it was recognised in the Madras Medical Board's reports previous to 1817. The fact is, our knowledge of the diffusion of cholera in India in earlier times is imperfect and limited, as our knowledge of the country was then imperfect and limited; but that it did prevail previous to 1817 appears undoubted, although perhaps called by other names. For instance, the word *modesh*, derived from *modna*, to tear, was adopted by the Portuguese, corrupted first into *mordexin*, and then into *mort-de-chien*, and often applied indiscriminately to the different forms of cholera.

It is true that some of the writings on which reliance is placed both in Europe and India to prove the antiquity of the disease, do not always describe the malady marked by suppression of urine, rice-water stools, and collapse, which in the present day is usually held to be true cholera. But this is only in accordance with the very numerous variations of the disease, as is sufficiently demonstrated by the number of names under which it has been described even in modern times, each term originating in some peculiar phase of the epidemic recorded. Thus we have in the writings of European authors the terms, Cholera asiatica; asphyxia algide; bilious; black; blue; dry; epidemic; endemic; febris remittens choleroidea; cholera foudroyante; malarious; malignant; nervous; nostras pestiferous; hæmorrhagica; serous; simplex; spasmodic; sporadic; syncopal tetanic. There are also cholerine; choleriform; choleroid disease; choleroid fever or sweating sickness; and the choleroid cholic of the Himalayas, which Webb regarded as a connecting link between cholera and colic. There is also reason to believe that cholera has been described as white dysentery, and as *fièvre pernicieuse*. Now all these are comparatively recent terms, called into use by the varying nature of the malady. The disease appeared under different phases, and

both in India and Europe was described as it appeared, and frequently under a different appellation.

There is indeed no disease in which the symptoms, although retaining certain characteristics, vary more, as is afterwards referred to under the head 'peculiar symptoms' (p. 119). The variability of the disease was noticed on the first reputed invasion of cholera into Europe in 1832, when Dr. Pennick [1] pointed out how the epidemic in England differed from the disease in Russia. About the same time Allan Webb [2] asked in India, 'Who can tell where cholera ends and plague begins,' or distinguish between certain forms of cholera and fever? But long before this Clarke [3] had recorded an epidemic on the Coromandel Coast presenting all the symptoms of cholera *except* purging. Macpherson states that the disease is essentially the same, but the cholera of one season varies from that of another. Ziemssen [4] remarks, 'Almost every epidemic has its physiognomy.' Sir Guyer Hunter [5] writes, 'As there are varying degrees of fever, so there are varying degrees of cholera. It may manifest itself by a slight looseness of the bowels, or as a sudden paralysis of the vaso-motor system.' Chevers [6] states, 'Familiar as I was with the cholera of Calcutta, the disease which I treated during a greater part of 1874 was of a type altogether new to me; no two cases were precisely the same;' and again, 'As the malarious poison produces all variations from a fatal remittent to the mildest ague, so with the cholera poison.' All this is merely in accordance with other diseases, many being undistinguishable by constant and invariable characteristics.

Mere degree is not sufficient ground for considering a disease different in essential character. There are mild and

[1] *Essay on Cholera*, 1832.
[2] *Pathologica Indica.*
[3] *Diseases of Hot Climates.*
[4] *Encyclopædia of Medicine.*
[5] *Report of Cholera in Egypt*, 1883.
[6] 'Notes on Indian Diseases,' *Med. Times*, 1883.

severe degrees of catarrh, influenza, scarlet fever, diphtheria, typhoid, &c., some so mild as to be scarcely recognisable. But it has been the custom to regard cholera as a disease marked by certain signs only, and which spreads epidemically, and to say a malady is not cholera when all these signs are not present, or even if it does not spread when they are present. This view I am not prepared to endorse. The variability of the types of numerous other diseases is fully recognised. No one would deny a particular case to be one of diphtheria, or typhoid, or measles, because the symptoms were mild, or because all the symptoms of a typical case were not presented, and it is only reasonable that the same breadth of view should be accorded to cholera. Then it would be recognised as what it really is, viz. a disease occurring at all times and in all countries, although not with the same degree of violence.

Passing from the *questio vexata* of the antiquity of cholera, it is admitted that in 1817 a great epidemic occurred at Jessore, in India. There is, however, reason to believe that previously to the time Jessore was stricken, cholera prevailed in the neighbourhood, particularly to the north. This Indian epidemic of 1817 is presumed to have spread to Ceylon, Burmah, and China. The next epidemic in Bengal, in 1826, is said to have spread to Cabul, Persia, Asiatic Russia, some parts of Europe and America. Those who doubt the antiquity of cholera in Europe regard this as its first appearance in the West. In 1840 another epidemic is supposed to have spread through Burmah to China, through Cabul to Russia in 1845, reaching Europe generally in 1848. But this 1840 epidemic is by another account (Macnamara) supposed to have been imported by Indian troops into China, spreading during 1841–42 *down* into Burmah, and in 1844 from Bokhara to Peshawur. Then it appeared at Lahore, sending off, as Dr. Arnott related, ramifications *down* the Sutledge and Indus to Sukkur. ‘ It continued its course down the

rivers . . . . and broke out at Hyderabad . . . . and afterwards proceeded onwards to Tatta and Kurrachee.' In 1849 it is supposed to have again spread from Bengal, reaching Egypt, Europe, and America in 1853–54. In 1860–61–62 it is surmised to have spread from Bengal to Mecca and Egypt, and so on to Europe and America in 1864–65. In 1883 it was presumed to have spread from India to Egypt, but this was proved erroneous by Sir Guyer Hunter, who demonstrated the previous existence of cholera in Egypt. And so, probably, would previous reported disseminations of the disease be proved erroneous could the facts be subjected to a similar critical investigation. For instance, in 1884 cholera was supposed to have spread to Marseilles, Toulon, &c.; but it is now known that cases of cholera occurred at least in Marseilles before 1884.[1] In 1885 it appeared in Spain; but judging from analogy and from the desire of the Spaniards to deny the presence of the malady, it is reasonable to infer Spain was not free in former years. In fact, as Cuningham[2] even admits, 'isolated cases of cholera occur in all countries every year.' Recently it has broken out again at Brindisi without any evidence of importation.

Habitat of cholera.—Although cholera in some one of its various phases frequently occurs in every country, it has a special preference for India, and especially for certain parts of India, as the Delta of Bengal, and the larger cities—Calcutta, Madras, Bombay, &c. This is sufficiently evident from the disease being always more or less prevalent in the localities named, and by the periodical outbreak of great epidemics. But, as Macnamara points out, all places visited by epidemics of intensity have usually features in common, viz., overcrowding, low-lying situation on alluvial soil, such as at the mouths of rivers, defective sanitation, or are malarious.

[1] Lucas, *Lancet*, 1884.

[2] *Cholera: What can the State do to Prevent*

Notwithstanding the so-called cholera of fowls (characterised by hæmorrhages, inflammation of the duodenum, sleeplessness, and the presence of bacilli in the blood); notwithstanding a report by Dr. Fairbrother,[1] regarding cholera in cats, at Delhi, in 1876; notwithstanding an alleged production of cholera in pigs by Vincent Richards in 1884;[2] and notwithstanding it is reported that Rictsh and Incall succeeded in producing cholera in dogs by injecting cultures of bacilli; notwithstanding Dr. Sicard, of Marseilles, states in 1854–55 birds succumbed in great numbers to the disease; notwithstanding horses have been said to suffer during a cholera epidemic (in Edinburgh in 1832, and at Lucknow in 1872); notwithstanding Dr. Murray thinks his dog was affected in 1864 at Agra, I still doubt if an animal cholera has been undoubtedly demonstrated. Cholera is essentially a disease present where men live. We are ignorant of the occurrence of cholera apart from human beings. Hence, as has been alleged, the ordinary spread of cholera along the lines of human intercourse by the great rivers and roads of India, and, as some believe, by the railroads in recent days. But Cunningham,[3] adopting Bryden's theory (referred to at p. 97), states, 'Want of easy communication does not retard cholera, and rapid means of communication does not accelerate it.' He holds that railways have no influence on the distribution of cholera, and that when men travel faster than cholera railways only bring people suffering from cholera. He states that in Bengal epidemics always move upwards in a general definite direction, and that such a thing as an epidemic moving southwards is absolutely unknown. Hence he holds that cholera is not due to human intercourse. However this may be, and the subject is afterwards referred to (p. 102), the fact remains that the *habitat* of cholera is where human beings live. Geological conditions do not

[1] *Lancet*, 1876. [2] *Ind. Med. Gaz.*, 1884.
[3] *Cholera: What can the State do to Prevent it?* 1885.

appear to exert much influence on the *habitat* of the disease. It is generally held that the soil favourable to cholera is a damp one, impregnated with organic matter, a condition in which it is especially exposed to the action of air as well as of water. The Cholera Commission of 1854 went so far as to state the disease only occurred in places situated on porous soil, permeable to air and water, and in which the latter was found at a moderate depth. But I have seen cholera on the granite rocks of Mount Aboo, on the black soil of Meywar, and on the sandy wastes of Marwar. Still, I believe the influence of an alluvial soil is favourable, not from the soil but from the dampness of such porous soil.

In most countries it is found there are *localities more or less practically exempt from cholera.* A few such localities were noted in India by Balfour,[1] who especially remarked on the freedom of laterite formations from the disease. Pondicherry is a locality which was long free from the disease. Marston[2] remarks, 'The Hurdwar pilgrims carried cholera with them into Mooltan in 1867, and as Mooltan had become a great railway terminus, it was alleged that its immunity from cholera—an immunity lasting over half a century—would be destroyed. But from 1868 to 1875, out of a population exceeding a million in Mooltan, Mozuffargarh and Dera Ghazi Khan, the districts tapped by railway communications, only seventeen deaths from cholera were registered, notwithstanding that the Punjab had suffered from no less than three epidemics during that interval. Cuningham[3] mentions Mussoorie and the Andaman Islands as places where cholera is practically unknown. Chevers,[4] commenting on such immunity, observes that such localities appear to have good natural drainage, and to be less noto-

[1] Balfour *On Localities in India exempt from Cholera.*
[2] Marston, *Lancet*, Sept., 1884.
[3] *Cholera: What can the State do to Prevent it?*
[4] *Ind. An. Med. Sci.*, vol. viii.

rious for bad conservancy and the neglect of sanitary regulations than other places; but this scarcely applies to all the localities mentioned above. In England, Birmingham not suffering in 1849, when the surrounding mining districts were decimated, has been attributed to good natural and artificial drainage on sandstone formation. This I know personally, having, in 1849, after some experience of cholera in the mining districts, been appointed to the Queen's Hospital, Birmingham, in anticipation of a cholera epidemic, which did not occur. The absence of the disease from the cider districts in England, although epidemic in adjoining districts, was considered by Headland and others due to the prophylactic influence of the vegetable acids so generally consumed; but in the wine-producing districts of Europe, where similar vegetable acids are consumed, there has been no similar immunity. On the Continent the cholera has presented at Stockholm several times, but never advanced so far north as Fahlun, and in the absence of geographical or geological lessons it was theorised that the quantity of sulphurous acid emitted into the atmosphere by the copper mines was the protective agency. It is also worth mentioning that the immunity which has been doubtless enjoyed by workers in certain gunpowder factories has been also attributed to the same acid. But unfortunately for such theories, while local outbreaks are a prominent feature of cholera, immunity has often been noticed of establishments where there was no trace of sulphurous acid. Lyons is another locality which, according to Pettenkofer,[1] has enjoyed a freedom from cholera, which is of course referred to by that authority as exemplifying the correctness of his ground-water theory. I, however, believe that immunity from cholera of any locality is only accidental, and that at some future time such locality may be attacked.

Elevation has an undoubted influence on cholera. Hirsch

[1] *Lancet*, Nov. 15, 1884.

stated there is no place in Germany, Italy, or Switzerland where cholera has prevailed at a height of 2,300 feet, and yet there is a considerable population at that level. The same author collected a number of examples, showing the adverse influence of low level. Farr made the statement that the number of attacks and the mortality of cholera stand in an inverse ratio to the elevation. As in Europe, so in India. The elevated plateaux and the hill stations although not exempt are comparatively so, disease rarely occurring there, and still more rarely becoming epidemic. Instances, however, have occurred in the Indian hill stations of epidemic cholera, notably under my observation, at Mount Aboo, with an elevation of 4,000 feet (clearly imported from the plains); at Muree, elevation 7,330 feet; at Dharmsala, elevation 6,000 feet; and at Kussowlee, which is nearly 6,000 feet high. In 1875 cases of cholera occurred almost simultaneously at the hill stations at Dharmsala, Simla, and Kasauli, and at the same time as in the Deccan and the Berars. But at Mahableswar and on the Neilgherries, both *inter-tropical* mountains, cholera has never prevailed epidemically. Elevation, it must be recollected, signifies comparative coolness and comparative purity of air, both conditions, especially in a tropical climate, conducive to good general health. The established fact that lowness of site conduces to the presence of cholera is regarded by Chapman[1] as offering a more intelligible explanation of its frequency along the borders of rivers and on the main lines of human traffic than does the allegation that cholera is propagated along these lines by human influence.

The incidence of cholera is usually severe in the most unsanitary localities, and, as a rule, such localities are the lowest. Particular spots, and even particular houses, in unsanitary localities have become locally infamous for cholera,

[1] *Cholera Curable*, 1885.

so much so that the disease has been attributed to local rather than to general causes. Experience has long ago established that in proportion as the air we breathe is pure it conduces to health; and, as Pettenkofer observes, if people with dirty surroundings suffer most from cholera, the explanation may possibly be that they suffer also in health from other epidemics, and even when there is no epidemic malady prevailing. But there are exceptions to the rule of cholera being most severe in the most unsanitary localities, and one is in the account of the Naples epidemic of 1884, of which I find the following statement:[1] 'The narrow, ill-ventilated streets intersecting blocks of buildings, in which the population teems like rabbits, have been precisely those in which cholera has numbered least victims; while the dwellings which, from their amplitude and situation, the freely aired thoroughfares dividing them, are in marked contrast to the preceding, have contributed the heaviest contingent to the hospital and the cemetery.' This is undoubtedly an unusual phase in the development of the disease in cities; but, from what we know of its severity in the most cleanly and well-situated barracks in India, it cannot be denied that it may occur in the best localities.

Influence of season.—In Europe cholera, whether *epidemic* or *nostras*, is, like diarrhœa, usually most prevalent during the hot autumnal weather. When it has occurred in Europe after the autumn the temperature has been higher than it is ordinarily at the time. In Paris, during the epidemic of November 1884, this was particularly noticed. In India, although cholera may occur at all seasons, it has a special preference for certain periods. Cuningham says there are two periods of maximum prevalence, viz., the spring months—March, April, May; and the winter months—November, December, and January. But there is a greater reason for stating that the maximum prevalence is usually

[1] *Lancet*, Nov. 1, 1884.

from the beginning of May till the end of October, comprising the hot weather and the rainy season. This is according to the observation of many independent observers, and is proved by an elaborate examination, instituted by Surgeon-General Murray, of the Indian sanitary statistics from 1866 to 1882. In all the provinces of India cholera reaches its maximum in July, excepting when interfered with by more than ordinarily warm winter seasons, as was the case in Bombay so recently as 1885, when by far the larger number of cases occurred during an exceptionally warm February; or when interfered with by scarcity or famine—which does not appear to have been taken into account by Cuningham—as was the case in Rajpootana, in Madras, and in Bombay in some of the years during the period referred to; and in Bengal when interfered with by the effects of a cyclone and inundation. Then the distress and want entailed rendered the seasonal force of cholera scarcely apparent; the people were predisposed to the disease, and the mortality was as great in the cold season as in any part of the hot or rainy season. So great in ordinary years is this season-prevalence of the malady, that Pettenkofer [1] speaks of it as the 'periodicity of cholera.'

Classes most liable to cholera.—It has already been stated that the incidence of cholera is usually greatest in low-lying unsanitary localities, and therefore those residing in such localities are ordinarily most subject to the disease. Of those living under fair sanitary conditions, unmarried soldiers are more liable than their married comrades, a fact, perhaps, explainable by the more irregular habits of life of the former, and by their more frequently visiting the native bazaars. According to Bryden, among the garrisons and prisons of India the liability of Europeans is more than ten times greater than that of natives. But it is doubtful if this is the case among the better classes of Europeans

[1] *Lancet*, Nov. 22, 1884.

in India. Among natives, Mussulmans suffer more than Hindus, and the Parsees, generally a wealthy class, suffer least. The Jews in Bombay enjoy none of the exemption witnessed among this class in the London epidemics, especially of 1849. Goorkhas have been supposed to be particularly liable to cholera, but the same may be said of all inhabitants of elevated or mountainous districts when brought down into the plains. Recruits have been observed to be especially liable to cholera, but so are all new comers into localities where cholera exists. Chevers states, 'The gravest and speediest incidence of cholera is on those recently arrived.' It is believed that men are proportionably more liable than women, excepting pregnant females, who seem specially prone. This liability of males is regarded by Chapman as proof of his theory of cholera being a disease of the nervous system, there being a much greater tendency to suffer from diseases of the nervous system having a fatal termination in males than in females, owing to the larger brain of the former. The incidence of cholera is also heavy on young children, proportionally more children suffering than in other epidemics. But infants enjoy a comparative immunity, and the fact has been frequently noted of infants sucking women with cholera until secretion of milk ceased without becoming affected. On the other hand, cholera has been noted in a newly born infant, the mother not suffering;[1] otherwise age exerts little influence. Persons who have suffered from cholera are rarely attacked again during the same epidemic, second attacks occurring in only ·08 per cent. of cases, but a previous attack confers no exemption in future epidemics, a fact altogether opposed to the alleged value of Ferran's cholera inoculations.

Intemperance must be ranked as a predisposing cause. It has been noted in European countries that during cholera epidemics more attacks occurred during the days immediately

[1] *Medical Times*, 1884.

after wages were paid, when the men had the means of dissipation. Brandy-drinkers are said to be the most liable (Pettenkofer). But any kind of alcohol has a special affinity for the nervous centres, and alcohol can itself excite purging and vomiting, and therefore appears particularly calculated to produce an especial predisposition to a disease of which nervous prostration, purging, and vomiting are the essential characteristics.

Fatigue is a still more important predisposing agent, which may, perhaps, explain the fact previously mentioned that new comers into an infected locality are very liable to cholera, as, especially in India, they have probably gone through a fatiguing journey. It also tends to explain the spread of cholera in India being so frequently connected with travelling by road. This has been demonstrated too frequently during the march of regiments, cholera being (at least, some years back) the rule rather than the exception whenever troops were moved in Lower Bengal. Maclean[1] attributed this to another factor as well as fatigue, advancing that the men were unable to bear the expenses of a march accompanied by their families and to supply themselves with sufficient food. The marches of French soldiers in Algeria have also been regarded as a predisposing cause of cholera. The influence of fatigue, combined with poor diet and other debilitating agencies, is shown in the frequent occurrence of cholera among pilgrims travelling from the great Hindoo shrines to their homes. They do not ordinarily suffer on the outward journey, but when to the fatigue of that journey, perhaps of hundreds of miles on foot, are added prolonged ceremonials, bathing several times daily and allowing their clothing to dry on them, exposure to the hot sun by day and the cold of night, without even tents, and probably insufficiency of food from want of means—when further debilitated by such causes they commence their homeward journey

[1] *Royal Sanitary Commission Blue Book.*

they ordinarily suffer from cholera *en route*. This was never more painfully demonstrated than after the great Hurdwar festival of 1867, of which we have the fact elaborately investigated by Surg.-General Murray of cholera accompanying the pilgrims on their homeward route in all directions.

Even when the influence of fatigue is not apparent, those subject to *want and famine* are always liable to cholera. The sanitary statistics previously referred to (p. 82) prove this; the influence of want and famine in India being sufficiently potent to override the influence of season, with the result of an even mortality from the disease for the whole year.

Fear or *panic* is undoubtedly a predisposing cause. In most great epidemics the number of attacks is largest and the mortality highest at first. This, perhaps, is susceptible of explanation by fear of the disease lessening as people become accustomed to hear of its ravages. But especially at the origin people are apt to forget that, even when cholera is at its worst, exemption from it is the rule rather than attack by it. It is probable that a certain amount of immunity has been enjoyed by those who have not feared infection. In the hospitals and among the cholera-stricken, medical officers and nurses have gone about their duty with no greater mortality than those who have kept aloof. Any *sudden shock*, which often causes an action of the bowels in good health, will render a person more liable to cholera. *Worry and anxiety* (which may even cause a chronic looseness of the bowels) will also predispose. Chapman [1] regards the well-known influence of fear as proof of cholera being a nervous disease, stating that fear exerts no such influence on the progress and termination of those diseases which there are valid reasons for believing to be results of blood-poisoning, viz., typhoid fever, scarlet fever, or small-pox. However this may be, observations prove that those persons who are

[1] *Cholera Curable*, 1885.

depressed or alarmed are most likely to become victims of cholera, that in the presence of a cholera epidemic panic intensifies its force, and in many instances apparently develops simple diarrhœa into cholera.

Bad food and eating to excess have often apparently converted a tendency to cholera into reality. It has been frequently observed that cholera seizures appear especially frequent in natives in India after a full meal. Bad fish, bad shell-fish, and bad pickled pork are known in several cases to have been followed by violent attacks with all the symptoms of cholera. Oysters have an especially bad name in this respect. Carpenter mentions an outbreak of cholera and choleraic diarrhœa among a number of school children who had eaten plentifully of spoiled oysters, and by which eleven lost their lives. In Bombay choleraic attacks after eating oysters are often heard of.

The older writers attributed the malady to fatigue, want, unwholesome ingesta, or bad food, chills, &c. They did not look for any specific poison, and they would not have believed what has since been asserted, 'that no amount of bad food or climatic influences have up to the present time induced an epidemic of cholera.' Some years back Dr. Tytler attempted to show that eating raw or diseased rice was the cause of cholera. Very recently the assertion has been revived that there is a connection between eating fish with worms in them and cholera. Several kinds of fish consumed in Bombay, as bummalos and prawns, are known at certain seasons of the year to be infested with worms. Indeed, it was reported that Balfour, in Bombay, in 1883, found in 'bummalos' bacterial organisms very similar to those discovered by Koch in cholera excreta.

The fish question has, however, been investigated by Surg.-Gen. Turnell, Madras, who describes a large number of parasites as occurring in fish, but sees no reason to connect them with cholera, although he does with worms. It

should, however, be recollected that a fish which is quite wholesome when absolutely fresh, may become otherwise if kept only a few hours in a moist hot atmosphere, from the formation of a ptomaine, which Anrep has shown produces semi-choleraic symptoms in dogs.

Next we have cholera attributed to *lunar influences*, to *epidemic constitutions* of the air, to 'une constitution médicale cholérique,' all being, more or less, relics of the ideas which in the middle ages attributed epidemics to comets, earthquakes, volcanoes, and sidereal changes; magnetism or electricity taking the place of Saturn or Mercury in the scheme of causation. The theory that cholera has its origin in some peculiar constitution of the air appears to have been propounded by the French physician, Aroga, who afterwards concluded the absence of ozone to be the *fons et origo mali*. Faraday asserted that this absence of ozone is connected with a state of negative electricity, which would always be found associated with cholera. Herepath, refining on this theory, believed that in Asiatic cholera the stomach is strongly electro-negative, the gastric mucous membrane secreting a free alkali; and hence as a curative agency recommended sulphuric acid. Others, again, have sought to prove that epidemics of cholera are always preceded or accompanied by some extraordinary meteorological change, which is indeed often the case. Thus Sir Guyer Hunter states that previous to the Egyptian epidemic of 1883 there was a still cloudless atmosphere, saturated air, and a peculiar yellow haze called by the Arabs *el hawa el asfar*, and it was noticed that sparrows deserted the locality where the haze appeared. Dr. Kirker's Egyptian meteorological records undoubtedly show that in June, 1883, in Egypt, as compared with thirteen previous Junes, the mean barometric pressure was somewhat below, and the mean temperature somewhat above the average. The maximum relative humidity was also highest, while ozone and wind force were low. From

the 12th to the 20th—cholera appearing in the middle of June—there was a period of excessive relative humidity, with occasional stagnancy of air. In the Crimea the outbreak of the disease seems to have been coincident with change from a dry to a humid atmosphere; at Agra, in 1884, with dry, east wind. Others have associated cholera with fogs and mists in which extraordinary developments of insect life have been observed. Bellew, in his recent 'History of Cholera in India from 1862 to 1881,' regards it as an intestinal catarrh produced by atmospheric conditions so strictly analogous to influenza or epidemic catarrh that, *mutatis mutandis*, the statement of the etiology and pathology of one applies to the other. Still no special meteorological phenomena can be regarded as the cause of or essential to cholera, for while peculiar atmospheric conditions have frequently been noticed coincident with the disease, it has often occurred when no such conditions were noted, although meteorological observations were conducted on the spot. On the other hand, such conditions have presented without cholera. This has caused recent writers to recur to epidemic influences as the cause. Thus Cunningham [1] says, 'Epidemic influence is a necessary factor even with contagionists. What it is we do not know, but of its existence there can be no question, because its effects are manifest.' And he, regarding epidemic influences as atmospheric changes, fancifully compares them to showers of rain 'which do not fall equally,' or to storms which do not prevail equally, thus accounting for the irregular distribution of cholera in its endemic area.

Others have theorised in a different manner. Limitation of the areas of cholera is sometimes singularly abrupt. It has been known to prevail on one side of a street, or a camp, or a town, or even in one part of a ship, or one wing of a building may be ravaged and the other escape. It may appear sud-

[1] *Cholera: What can the State do to Prevent it?*

denly, and as suddenly disappear. It has burst out in every regiment at a station, in other places it has only attacked one regiment. Now we have experience of other disturbances which are localised, which appear suddenly and then disappear in the small whirlwinds so frequently seen in India, especially in the hot weather, varying in diameter from a foot to many feet, and varying also in force. But if it were not for dust, leaves, and other light substances carried round with the whirl, we should not know what is occurring. Dr. Bonavia[1] says, 'If dynamic disturbances of such limited area are possible, it is not impossible that magneto-electric disturbances of a larger area, but quite limited, should be sometimes the exciting cause of cholera.' And the idea is strengthened by the facts that the most certain means of avoiding cholera is to move from the locality, that is to get out of the cholera cyclone; also that the sudden appearance and disappearance of the disease and the limitation of area is not compatible with the poison theory.

As regards the influence of *rainfall*, the records of cholera show numerous instances when the disease commenced after a heavy fall, and numerous examples when it ceased or lessened after a heavy fall. The fact is, more importance has been attached to special meteorological conditions than they deserve. At the same time, it may be admitted that as a high moist temperature favours putrefaction and augments the impurity of the air, it also probably aids on the transmission of germs.

The fact appears to be that great heat, wide ranges of temperature, disturbances of atmospheric electricity, or great magnetic disturbances (usually present when the largest sun-spots are seen), abnormal falls of rain, are all more or less predisposing agencies; although none are decidedly necessary as exciting causes.

Other *predisposing* causes are nocturnal influences,

[1] *Ind. Med. Gaz.*, Feb., 1876.

vitality being lowest during the morning hours of sleep; the incautious use of purgative medicines, and dentition. Insanity has also been regarded as a predisposing cause, but this is scarcely in accordance with the experience of Indian lunatic asylums.

Allan Webb, in his 'Pathologica Indica,' attributed cholera to sulphuretted hydrogen, which he stated was emitted from the earth in various parts of Western India. Kirke attributed cholera, and also fever, to mephitic vapours, chiefly sulphuretted hydrogen, emitted from the rocks at Sukkur. Dickson saw in it the same disease described by Travers as 'constitutional irritation.' Gaffney[1] regards it as a form of blood-poisoning from decomposing urine, by which urea is introduced in other forms into the blood. Beaman regarded deficiency of salt as the cause, alleging the salt monopoly in India caused cholera, and recommending as a cure salt in water, to cause vomiting. Gull and Copland taught that cholera was a profound depression or diminution of the vital force of the sympathetic. Chapman,[2] with great ability, reasons that all the symptoms are due to a simultaneous and abnormal superabundance of blood in, and excessive preternatural activity of, both the spinal cord and sympathetic nervous centres, brought on by heat, and as a curative agent Chapman extols the spinal ice-bag. Others have seen in cholera the same disease as insolation.

Next we have cholera attributed to malarious influences, a theory which has found many advocates. Under this head it is worth recalling the ancient Sanskrit term for cholera, *vishuchuka* or *bishuchuka* (mentioned at p. 72), which means fever and incessant purging, thus coupling the two maladies together. McCulloch, so far back as 1827, wrote, 'Cholera belongs predominantly, if not exclusively, to the same climates, the same soils, or generally to all those countries in which other diseases of malaria abound,' and afterwards he remarks

[1] *Ind. Med. Gaz.*, Jan., 1884. *Curable*, 1884.

that the disease is common where fevers are common, and rare where fevers are rare. Since that period the theory of the affinity of cholera and ague has found many able supporters, who have all advanced much the same arguments first promulgated by McCulloch. Thus Inspector-General Munro[1] calls cholera and malarious fever one and the same disease. A disease described as the choleroid colic of the Himalayas was regarded as the connecting link between fever and cholera.[2] Macpherson[3] states *fièvre pernicieuse* has always appeared to resemble cholera. Dr. Curran[4] has published observations on the identity displayed by the cholera and fever of Peshawur. Bellew[5] states the Peshawur fever is exactly like cholera. Surgeon-Major Green[6] has recorded cases commencing as ague and terminating as cholera. Professor Blanc[7] has published similar instances commencing with fever and ending as cholera, to which this able observer applies the term 'malarious cholera.' Sullivan[8] observes in tropical climates 'the cold stage of cholera may be mistaken for an attack of algide pernicious fever.' Macnamara[9] writes, 'The epidemics of Northern India show how constantly those of cholera and malarious fever are associated, and tends to identity of cause.' Sutherland[10] asserts malaria is concerned in the prevalence of cholera. Chevers,[11] than whom no authority is entitled to greater respect, observes, 'Several forms of fever have the appearance of forming a continuous chain of clear intermediate etiological links between malarious remittent and Asiatic cholera.' Professor Waters[12] has noticed attacks, which at the commencement were dis-

[1] *Army Med. Report*, 1872.
[2] *Pathologica Indica.*
[3] *Annals of Cholera.*
[4] *Ind. An. Med. Science*, July, 1876.
[5] *Ind. Med. Gaz.*, 1871.
[6] *Ibid.*, May, 1880.
[7] *Medical Times*, Aug. 1883.
[8] *Diseases of Tropical Climates.*
[9] *Himalayan India.*
[10] *Report of the Royal Sanitary Commission.*
[11] *Medical Times*, 1883.
[12] *Epidemics: a Lecture delivered at the Sassoon Institute, Bombay*, 1884.

tinctly choleraic, quickly give place to intense fever followed by profuse perspiration. Dr. Smith, A.M.D., wrote a volume[1] to show how strong proofs these are that 'cholera is a member of the group of febrile diseases expressed in the highest degree of intensity,' and he quotes the reports of various medical officers to the same effect. Dr. Smith argues that both diseases occur at the same season; that bad fever years are usually bad cholera years, that in both there is an initial period of malaise, that in severe fever there is pain of limbs almost amounting to cramp, blueness and collapse, and although there may be no suppression of urine, he regards this as merely an indication of less intensity. Smith also observes that the intestines found affected in fever are the same as those implicated in cholera, the lesions only differing in degree. Similar observations are forthcoming from the West Indies. Sullivan states that in the gastro-intestinal nervous spasmodic or adynamic form of yellow fever, the patient appears struck with horror, stupefied, and paralysed. He next complains of abdominal and cardiac pain, and is bathed in cold sweat. Respiration becomes affected, he looks dark and livid, and after passing a motion falls down lifeless.

Others, while not regarding cholera and fever as the same disease, have admitted a connection. Thus Parkes stated in malarious places cholera assumes a periodical character. Lewis and Cunningham give reasons for holding cholera to be a malarious disease, or in other words, that 'the primary cause is a telluric or soil-begotten poison like malaria, elaborated and applied without the intervention of man.'[2] Pettenkofer states, as it is the result of the poison which springs from the soil, it is in this sense a miasmatic disease, just as much as marsh fever. But Ziemssen observes, intermittent fever has no particular relation to cholera, though both stand

[1] *Fever and Cholera from a New Point of View.*

[2] *Thirteenth Report of the Sanitary Commissioner with the Government of India.*

in connection with the degree of the humidity of the soil. Other observers have also stated their disbelief in any connection between cholera and fever. Sircar[1] has never observed any connection between fever and cholera, and thinks they are distinct diseases. Macpherson also points out that cholera is not ushered in by rigors. Lawson some time back published an able paper 'On the Influence of Epidemics of Fever in *Checking* the Advance of Cholera.' Systematic writers always describe cholera and fever as different maladies. Probably the similarity which has been noted by so many is due to the fact of cholera, like most other diseases in a malarious country, assuming a febrile character. A typical case of cholera and a typical case of fever are certainly sufficiently distinct, presenting less features in common than many other different diseases. As Inspector-General Murray observed of the cholera of Central India in 1860, 'Many of the cases assume an intermittent type,' and this appears to be the principal feature of similarity. The algidity of fever which shows itself insidiously in the middle of an attack of intermittent, and which is characterised by pale and sharpened features, lips drawn in and blue, eyes hollow and pupils dilated, is not the algidity of cholera.

Next there is the *fœcal theory*, early conceived and very generally accepted. Under this idea cholera dejections were regarded as poisonous if introduced into the system. But it soon became evident that as filth alone did not always induce cholera, so the evacuations of the cholera-stricken did not always appear to be poisonous. The immunity of night-soil men was noticed, who did not seem more liable to cholera than other classes. Medical men and others had their clothes saturated with cholera dejecta, yet escaped the disease. Direct experiment with cholera fæces by Majendie, Lindsay, Mayer, and at a later date by Lewis and Cunningham, V. Richards, and others in India yielded negative results. Chevers re-

[1] *Ind. Med. Gaz.*, 1880.

cently states, 'No one has proved to my satisfaction that cholera stools contain a poison capable of producing cholera in those who swallow them.' It therefore became necessary to modify conclusions, which was done by Thirsch and Pettenkofer, who theorised some change in cholera evacuations taking place without the body vitalised the poison which is again taken into the system. At first it was presumed this might depend on putrefaction, but after a time it began to be believed that, as Ziemssen states, putrefaction rather diminishes the capacity for infection, and that the bacteria of decomposition destroy the poison of cholera. Although many ceased to think the evacuations the actual cause of cholera, they still regarded such discharges as the medium in which the poison passed through one stage of development, a stage perhaps immediately antecedent to that of putrefaction. C. Macnamara relates an instance strongly corroborative. Some rice-water cholera evacuations were mixed with water, and stood exposed to the sun for some hours. Accidentally 19 persons drank of this, and five were attacked by cholera within 36 hours.

With reference to the above it may be noted that Maurin and Lange say on the fourth day a mucus appears on cholera stools which discharges spores, from which, if in contact with organic matter, are developed the bacillus of Koch.

The theory vitalising the poison outside the body necessitated the search for some nidus in which it might develop. This was forthcoming in Pettenkofer's ground-water theory, which is an elaboration of Sir J. Pringle's[1] idea, who stated, 'In Dutch Brabant the people are more or less subject to intermittent, in proportion to the distance of the water from the surface, so that by looking into their wells one may form a judgment of the comparative healthiness of villages.' Pettenkofer's theory postulates the soil with being the laboratory in which the poison of cholera contained in

[1] *On the Diseases of the Army in Flanders*, 1780.

cholera stools is vitalised, and the air emitted from the soil as the vehicle by which the poison gains access to man. The process of vitalisation and diffusion are held to depend on several conditions, the most essential being variations of soil, moisture, and rapidity of escape of air. When the ground water is high the upper soil is much damper, and less of it pervious to air. Increased cholera accompanies a low state of ground water, and a rise of water is followed by a diminution of the disease. Recently, Pettenkofer[1] endeavoured to prove of Calcutta that the curve of the disease falls while that of rain rises. This does not, however, agree with the observations of those better acquainted with India. If the study of cholera were confined to India it would be easy to demonstrate the fallacy of Pettenkofer's theory. Lewis and Cunningham, indeed, tested the truth of it with negative results.[2] The simultaneous appearance of cholera with a fall of rain too limited to reach the subsoil which is often noticed in India, is altogether against Pettenkofer's ideas. So is the fact of cholera prevailing in the semi-desert districts—at Bickanir, for instance—where water is three, six, or even eight hundred feet from the surface, and where only four or five inches of rain falls annually. Pettenkofer goes too far when he maintains that a certain condition of soil and ground water is necessary for the development of cholera. The outbreak in the North Staffordshire Regiment at the foot of the Bolan Pass, in December 1884, is an instance utterly opposed to Pettenkofer's theory, the surface there being dry and rocky, the air pure from the mountains, and the water clear and pure.

The *fungoid theory*, which regarded some growth in the fæcal material as the cause of cholera, originated with Boehm in 1838, who attributed cholera to a cryptogamic growth found in the dejecta. In 1848 Parkes noted in cholera dejecta dark yellow granules and vibriones. Grove in 1849

[1] *Lancet*, Nov. 8, 1884. [2] *Ibid.*, Dec. 13, 1884.

noticed comma-shaped bodies which he regarded as fungoid. In 1849 Swayne and Brittain published drawings of 'cholera cells,' afterwards asserted by Busk to be spores introduced with the food. In the same year similar appearances were detected by Pouchet. Farr, in reporting on the English cholera of 1848–49, said it was induced by a specific cell which he called *cholerine*, and which he theorised had the property of multiplication in air, water, and food. Budd, then holding similar views, proposed destroying the fungus by chemical means. Hassal in 1853 detected something very similar to the comma bacillus. In 1854 Lindsay described globular corpuscles with nucleoli. In 1873 Hassal stated vibroines were always present in rice-water evacuations. Budd in 1873 announced similar bodies, classed by Willan and Busk as *uredo*. In 1866 Bristowe found something resembling the comma bacillus. Other formations have also been found by Klob (*leptothrix*), Thorne (*cylinder fungi*), and Hallier (*micrococci*). But Lewis and Cunningham in India in 1870–74 proved the non-existence of any specific cell, or other body peculiar to cholera, and not found in other dejecta. These observers explained the sources and figured the outline of all bodies observed, and state that on standing cholera stools deposit a finely granular whitish grey substance, containing epithelium, shreds of tissue, triple phosphates, bacteria, blood-corpuscles, salts of lime, common salt, and albumen.

The next researches on the subject are those of the German Commission in Egypt and India in 1883–84, under the superintendence of Koch. A bacillus shaped like the German comma was found in the intestinal walls chiefly in and around the follicles. Briefly stated, Koch's conclusions were, that the number of comma-shaped organisms in the intestinal canal is in proportion to the acuteness of the attack; that these organisms generate within the body a ferment or poison; that they are not found under any conditions other than

in connection with cholera; that their presence in a tank which supplied certain cholera-affected villages near Calcutta, was practically a proof of the causal connection between these organisms and the disease. But Drs. Klein and Gibbs, who were sent out to India in 1884 to investigate the matter, refuted these conclusions. And a commission, appointed by the Secretary of State for India to enquire into the conflicting statements made by Koch, and Klein, and Gibbs, reported that comma-shaped bacilli are ordinarily present in cholera dejections; that similar-shaped organisms are ordinarily present in different parts of the alimentary canal in health; that they are developed to an unusual extent in some diseases characterised by hyper-secretion of the intestines; that the bacilli ordinarily found in cholera do not induce the disease in the lower animals, and that there are no real grounds for assuming they do so in man.

The next theory worthy of notice is that of Bryden, who from a study of the different Indian epidemics drew the conclusions that a poison elaborated in Bengal spreads in a certain direction through the air, and is distributed on the surface of the ground, where it may be vitalised several times, continuing to spread, and arriving at certain times and at certain places with almost mathematical precision. After Surg.-Gen. Murray had elaborately traced the course of the cholera from the Hurdwar fair along the various routes taken by the returning pilgrims, Bryden, as the result of his studies, stated his opinion that the geographical distribution of the cholera would have been exactly the same had there been no Hurdwar epidemic. Bryden's views, however, did not find general acceptance; the theory which would regard cholera as an 'earth-born, air-conveyed son of mystery,' arriving at a certain time at a certain place, being contrary to facts, calculated to stay research, and likely to indicate to the ignorant masses the uselessness of sanitary precautions. It is Bryden's theory, on which Cuningham

bases his assertion (*vide* p. 77), that epidemics have a general definite direction. In remarking on the Hurdwar epidemic Pettenkofer recently stated, 'According to the contagionists, an outbreak ought to have taken place in every locality to which pilgrims wandered, but that really disease only occurred when time and place and local conditions were favourable.' I fail to see the force of this, as we know other maladies do not occur in every place where those affected present themselves.

The next recent theory is Gautier's, who has shown that in the dead body, and even in the living, ptomaines are formed, and he attributes cholera to a ptomaine. This does not, however, accord with the occurrence of cholera by communication, or explain why cholera should follow the arrival of a person from an infected district.

Although not discovered, both analogous reasoning and the last-mentioned characteristics of the malady lead to the inference that a specific poison must exist. Surg.-Gen. Cuningham, however, states 'The doctrine of germs or contagion will not account for Indian epidemics.' But until we isolate or identify the cholera poison, or define with precision the conditions on which the disease depends, all our reasoning must necessarily be imperfect and subject to doubt. We can only hope to establish different degrees of probability, or, as some may regard it, of moral certainty. Now that we can certainly trace every form of fermentation and putrefaction to the development of saprophytes, or minute bodies vegetating in decomposable organic matter, all the facts supporting the doctrine of zymosis, first introduced by the late W. Farr, go to strengthen the conception of germs. But as regards cholera all that can be said is that the present state of our knowledge tends to the belief that there is a poison acting primarily on the nervous system.

The theory of a poison being admitted, the questions of

origin and spread present. The agent having once originated, either it must be conveyed from place to place by human intercourse, or it must be conveyed through the atmosphere, or by water for an indefinite distance; or it must be the revitalisation of the quiescent germs of a previous epidemic; or it must originate *de novo*. As regards the *de novo* theory one section do not believe in such origin, arguing that as a seed, or a shrub, or a tree, or an animal must proceed from its like, so disease must emanate from disease. Another section do not see why what has once been formed should not be again produced. They argue that to deny *de novo* origin limits power to one effort of generation. The conditions which call disease-poisons into existence must be capable of precise repetition, and the cause being present the effect will follow. There is nothing inconsistent in the admission that what has originated under certain conditions may again originate under the same conditions. That animals and vegetables do not now originate *de novo* has been attributed[1] to the absence at the present time of the circumstances fitted to call forth these superior formations. But this is no reason why the circumstances fitted to call disease poisons into being should not exist, and why disease-poisons should not arise—as gases and malaria do. Something more than an organism is required to ensure produce. Unless one particle of matter is acted on by another the result is *nil*. Oxygen and hydrogen placed in juxtaposition at an ordinary temperature will not combine; flame being applied, force is evolved, with the result—water. Light or heat may be applied in any amount to an egg, a seed, or a dry *rotifer*, but they will not change if air be withheld from the first or moisture from the two latter. Given certain but unknown conditions of matter, certain but unknown conditions of atmosphere, and certain conditions of human constitution, and I fail to see why a disease-poison should

[1] Alison, *Lancet*, March, 1863.

not be produced and act, as we know the poison must have been once elaborated. Under this belief only do I think the irregular occurrence of cholera in India can be explained.

It is unfortunate the requirements and results cannot be stated with the algebraical precision once attempted;[1] or that we cannot be satisfied with the conclusions of one of the civilian sanitary commissioners, referred to at p. 4, who believed he had shown that cholera was 'something which spread in a roundabout manner.'[2]

It occurred to me to investigate several epidemics of cholera occurring in the extensive province of Rajpootana, and I could cite numerous instances, presenting in widely different localities, where the malady occurred under the impossibility of direct intercourse. I say the impossibility of direct intercourse, because the disease broke out at nearly the same time at places so far apart, between which, in a country destitute of railways or waterways, communication was impossible—in many cases impossible even by express rail service. The cholera therefore must have been conveyed through the atmosphere, or it must have been the revival of a previous epidemic, or, as most likely, it must have originated *de novo*. I do not deny that it may be conveyed through the atmosphere. Most persons who have been much in the East will recollect how rapidly and how far impalpable sand-dust is brought after probably a high wind, from the semi-desert sandy districts, travelling many miles in a night, sometimes, apparently, against the wind, and doubtless brought by an upper current, and in a calm spreading in all directions. The cholera germ, for aught we know, may be more impalpable than the finest sand, or even than the atoms composing the scent of a flower. If the invisible microphytes, which cause putrefaction, are wafted about as bacterial clouds, as stated by Pasteur and Tyndal,

[1] *Lancet*, August, 1869. [2] *Punjaub Cholera Report*, 1861.

there seems no reason why invisible or even more minute disease-germs should not be so wafted about. Influenza is a disease which spreads over provinces, and no one questions the theory which regards the atmosphere as the medium of communication, yet many authorities do not admit the spread of cholera in a similar manner. But it is consistent with reason, as well as with experience, that communicable diseases are liable to be communicated in different ways. In the present state of science it is not wise to affirm or deny communication in any one particular manner. It is quite possible that some contagia may be more easily transmitted by some media than by others, and it is not unlikely that differences of climate or season may affect transmission by different means and media. It does not necessarily follow that a poison or germ, capable of being conveyed by water, is not liable to be conveyed by air. Numberless instances of cholera might be quoted, in which entrance of the poison, *per* the respiratory tracts, seems to be the only explanation. Mons. Ramon de Luna, at a meeting of the *Académie de Sciences*, expressed his views that the poison acts exclusively through the respiratory passages, where it certainly would come into more intimate relations with the blood, spread out on the capillary network of the air-cells, than when introduced with food or drink into the alimentary canal.

An objection to the theory of germs spreading indefinitely through the atmosphere is that the oxygen of the air must tend to destroy them. A similar objection applies to the theory which would re-vitalise dormant germs of a previous epidemic. Although cases have been recorded[1] of both cholera and small-pox following the opening up of old graveyards, where persons dying of these diseases had been buried, I am not prepared to accept the re-vitalisation theory, as earth and air tend to the destruction or metamorphosis of matter rather than to its preservation. One may imagine

[1] Author's *Health in the Tropics.*

germs being preserved, if buried deep in an impervious clay soil; but I question the preservation, for an indefinite time, of such minute and fragile material when exposed to the action of oxygen, while floating in the atmosphere or on the surface of the earth.

At page 100 I state, 'I could cite numerous instances where the malady occurred under the impossibility of direct intercourse.' On the other hand, I could relate numerous other instances (commencing as far back as 1849 in England), where the conveyance of the disease by human intercourse appeared as certain as the exact sciences. The fact of people going from one house or one village where cholera prevailed, their arrival in another house or village being immediately followed by cholera in themselves and others, *cannot* be ignored; and of this I have recorded various examples.[1] These are what Cuningham would call 'little facts,'[2] which he regards as at variance with the general history of cholera, and therefore wrong. But little facts are as stubborn as great facts and, when instances of the kind present the inevitable conclusion of direct communication, *cannot* be ignored for the fanciful idea previously referred to, of an epidemic influence descending in an unequal and irregular manner like showers of rain (p. 88), or for the idea of cholera being a magnetic disturbance coming and going as a whirlwind (p. 89). Neither can these little facts be explained away on the theory of Chapman (p. 90). The three theories mentioned above would relegate to the class of coincidences all those numerous instances of cholera having immediately followed the arrival of a cholera-stricken person, or of a person from a cholera-stricken locality into an uninfected locality, which cannot be accepted. It has been proved that birds can sub-

[1] Author's *Rajpootana Dispensary, Jail, Vaccination and Sanitary Reports*, from 1868 to 1877. Published as selections from the *Records of the Government of India, For. Dep.*

[2] *Cholera: What can the State do to Prevent it?*

sist for a certain time on a certain amount of air—say, for instance, three hours on ten cubic feet—but if the bird be taken out at the end of two hours and replaced by another bird, the fresh one dies at once. Hence it is reasoned that animals, including human beings, can acquire a certain tolerance of a vitiated atmosphere. And it has been argued that the real cause of cholera first attacking persons arriving is that they came into a locality where cholera was to occur, and being least habituated to the conditions were attacked first. But it is not those who come from any one locality to any other who apparently spread cholera. It is those who come from a cholera locality into one not yet infected. So far as I am aware, there are no instances of persons coming from an uninfected locality into another uninfected locality, being seized with cholera immediately afterwards, and apparently spreading the disease.

The theory of water being the vehicle by which cholera is conveyed into the system has obtained the greatest number of supporters. In 1849 Dr. Snow first pointed out the probability of cholera being disseminated by drinking water contaminated with fæcal material. This was not very well received, for the London College of Physicians, in a report on the epidemic of 1848–49 (published 1854), considered Snow's theory untenable, and were of opinion that human intercourse was of most importance in propagating the disease. The College, however, remarked that it by no means follows that cholera is always propagated in one way. But in 1854 occurred the celebrated Broad Street pump case, which was investigated by a committee whose report contained evidence, convincing to many, of the origin of 200 cases from the transmission of the cholera poison from a child first attacked into the water. An occurrence adding much strength to the water theory was as follows. A Mr. Eley, having business in Broad Street, returned every evening to his house at Hounslow, taking with him for the use of his mother and

sister a bottle of Broad Street pump water. Both these ladies suffered from cholera, although there was no disease in their neighbourhood. Then Dr. Snow showed that in districts partly supplied by pure water from the Lambeth Company, and partly by impure water from Southwark, attacks of cholera occurred chiefly in houses supplied from the latter source. Snow's views were soon accepted wholly or partially by Budd, Acland, Carpenter, Allison, Routh, Sutherland, and many others. Numerous other apparently conclusive instances are on record of the conveyance of the disease by water. Thus in England there is the College Street case—where those drinking from Jacob's well were affected, into which well sewage was found to trickle from a cholera-infected house. In India in the Hooghly several P. & O. ships were moored close together. One vessel, having something wrong with the water tanks, was supplied with water by bheestees from the shore, whereupon twelve cases of cholera occurred, while the other ships remained free. De Renzy, in his report on one of the Punjaub epidemics, asserts a supply of pure water will remove the most fruitful cause. The International Sanitary Commission were unanimous in referring to water as the medium. Macnamara also believes water to be the principal, if not the sole medium of conveyance. Then there are numerous places in which a supply of pure water has been followed by exemption from or diminution of the disease. Bombay may be mentioned as a city in which cholera has been less constant since the wells were closed and a water-supply procured from lakes in the distant hills. Glasgow, Exeter, Hull, Moscow, are localities which have escaped recent epidemics, consequent, as it is believed, on the supply of better water. Aitkin observes the evidence of communicability by means of water has, since 1854, when first demonstrated by Snow, become almost overwhelming. Chapman, who regards cholera as a nervous disease, nevertheless states that of all avoidable causes of cholera impure

water is the most common and most baneful. He does not, however, regard water as containing a cholera poison, but he considers bad water acts on the terminal branches of the sensory nerves in the alimentary canal, in the same way as noxious effluvia do on the sensory nerves and the respiratory mucous membrane, exerting a profoundly depressing influence through the myriad paths of reflex action on an hyperæmic condition of the nervous centres, the effect of heat (*vide* p. 90).

On the other hand, Surgeon-General Cuningham asserts the water theory is negatived by the whole history of cholera, and in his sanitary report for 1872 he states that after careful examination of 100 centres, including regiments, jails, and villages, in no one instance could he discover that the disease was due to drinking water from any particular well. It is also mentioned that in a recent epidemic in Bombay (1883) the disease showed no preference between the minority who still use well water and the majority who are supplied from the waterworks.[1]

It is useless, however, multiplying instances when the disease was apparently due to impure water, and when no such cause could be ascribed. On both sides the cases which might be brought forward are legion. The weight of evidence is, however, undoubtedly in favour of the theory that water is often the medium by which the poison of cholera is conveyed. The practical lesson is that when a sudden attack of large numbers in the same locality occurs, the position distinctly points to community of cause, and the water should be looked to.

If it be admitted that the germs or poison of cholera may be conveyed by water, it may also be admitted that it may be conveyed by food, either by admixture with water or *per se*. Many instances have been recorded where food seems to have been the vehicle through which the poison was

[1] *Lancet*, Nov. 24, 1883.

conveyed into the system. Pettenkofer especially mentions watery, slimy food as having the property of attaching to itself cholera germs and of maintaining them in an active condition. The International Sanitary Conference of 1874 were unanimous in their opinion of food being a medium, and I believe the poison frequently is conveyed into food by means of flies. It is during the cholera season, or from the commencement to the end of the rains (*vide* p. 82) that myriads of flies abound in many parts of India, clustering on every article of food in the bazaars, where no attempt at protection is made; and some of these flies may have proceeded directly from a cholera stool. Further, I have watched people manufacturing food, and especially 'koftas' (a species of sausage eaten by Mahomedans), chopping up indiscriminately all the flies happening to alight on the food or knife. I have somewhere seen it stated that flies, like birds, especially swallows, desert cholera localities. I am certain flies do not, and, notwithstanding many recorded instances, the desertion by birds of cholera localities, particularly birds of prey, is exceptional.

Conveyance of cholera by material, such as clothing, rags, merchandise, is a disputed point. If it were thus conveyed, soiled clothing or rags would appear the most likely media. Pettenkofer states such conveyance is free from doubt, and the fact of washerwomen suffering has been noticed by others. Carpenter [1] says it is an established fact that linen soiled with cholera discharges may not only impart the germs to those who unfold it when fresh, but that after being dried and packed it may infect persons at any distance who unfold it. In India, however, we do not find this to be the case, 'dhobees' or washermen not being more liable to cholera than others. The International Cholera Conference of 1874 were, however, unanimously of opinion that the disease is transmitted by personal effects, although

[1] 'Zymotic Diseases,' *Nineteenth Century*, 1884.

not so unanimous as to its conveyance by merchandise. The most recent instructions from the Board of Trade (dt. Jan. 1886) proceed on the assumption that cholera may be conveyed by clothing, as they order articles soiled with cholera discharges to be destroyed, other articles used to be disinfected; and in the case of seamen dying of cholera at a foreign port, they instruct consuls to give directions for the destruction of the clothing, which is never to be sent to the United Kingdom.

I can refer to instances where the conveyance by clothing seemed clear, and to another when the conveyance by merchandise seemed equally clear. In 1885 cholera prevailed in the city and station of Dharwar. It suddenly broke out in the lunatic asylum. On inquiry it was found the bheestee's son, who lived a short distance outside, had suffered. The bheestee himself did not suffer, but as he passed in and out every day, the cholera must either have been introduced on his clothing, it must have originated within the asylum, or it must have been due to general atmospheric causes. The cleanliness of the asylum, the fact of others in the immediate locality not suffering, the freedom of the bheestee himself from disease, pointed to the only rational conclusion, viz., the conveyance of the disease by the clothing of the man.

There had been no cholera at Aden since the year 1867. The steamer 'Columbian' arrived at Aden from Bombay on July 31, 1881, having 650 pilgrims on board, and a cargo of rice packed in gunny bags which she commenced unloading. On arrival the steamer was visited by the port surgeon, and found in as good condition and as cleanly as pilgrim ships usually are. On the night of August 1, a coolie who had been employed during the day unloading rice was attacked with cholera. On August 2, two more of the working party were found suffering, and two more were sent on shore sick from the ship. On August 3, nine altogether had

been attacked, after which there were numerous cases. No case of cholera had occurred on board the 'Columbian' during the voyage from Bombay to Aden, but cholera prevailed at Bombay when the ship left. It was considered likely that the germs of the disease were secreted among the rice-bags in the hold, and liberated when the bags were removed by the coolies. Although only two coolies were actually attacked on board, it is probable the germs were received by all the nine coolies first attacked on August 1, as it is believed the incubation of cholera may extend from a few hours to ten days (*vide* p. 112). The Aden coolies, as a rule, are indifferently nourished, they were keeping the Ramazan fast, and therefore many would be working on an empty stomach. Their system would be more than ordinarily debilitated, and more than ordinarily predisposed to disease. It should also be mentioned that some of the workmen stated they noticed a bad smell in the hold.

It has already been stated that cholera was in Bombay when the 'Columbian' left, and it was thought probable that one or more of the rice-*bags* had been accidentally contaminated by cholera dejections, either before the rice was packed or on its passage from the merchant's stores to the vessel. Even one of the men engaged in shipping the rice might have vomited or passed choleraic discharge on a bag. The vitality of the germ thus communicated would be preserved by the absence of the great purifier—oxygen—amongst the densely packed rice-bags in the hold. The rapid removal of the tainted bags may have prevented infection among the pilgrims, who were not brought into immediate contact with the cargo.

Surgeon-General Cuningham, however, who in his capacity as Sanitary Commissioner with the Government of India commented on the Report of the Committee on the Aden Cholera, did not agree with the above conclusions. He remarked that for some time previously the death-rate at Aden

had been high, owing to an influx of persons from the interior suffering from want; that the localities in which the coolies live were in a very unsanitary condition; that bowel complaints are not uncommon during the Ramazan; that some of the persons first attacked had not been on board the 'Columbian' at all; that the coolies first attacked were seized so immediately that the attacks seem hardly explicable on the committee's theory (meaning, I presume, the germs had not time to act); that although 700 people came from Bombay to Aden on board the ship, no one was affected; that the ship pursued her voyage without recurrence of cholera; that the rice was consumed at Aden, producing no cholera; that the conclusion of the committee is not in accordance with the experience of Aden, where for many years traffic with Bombay has been constant and enormous, without the development of cholera at the former port. Surgeon-General Cuningham therefore regarded the occurrence of cholera at Aden, and the arrival of the pilgrim ship as simply a coincidence in point of time.

Nevertheless, having investigated the circumstances carefully on the spot, I still believe the cholera of Aden in 1881 was introduced by the 'Columbian.' With reference to Surgeon-General Cuningham's objection, I observe that a high death-rate among coolies living in unsanitary localities, and frequency of bowel complaints are not uncommon at Aden, but do not generally develope cholera; that the persons first attacked had been on board the 'Columbian,' although very soon others were attacked who had not been on board; that there is reason to believe the incubation of cholera may be only a few hours (*vide* p. 112); that it was not until the rice-bags were liberated that anyone became affected; that with the removal of the rice-bags the danger was removed from the ship; that the gunny-bag and *not* the rice was contaminated; that because merchandise is landed 99 times without conveying disease, is no reason why

disease cannot be conveyed on the 100th; that if merely a coincidence in point of time, it is very strange the disease should have developed in the ship coolies instead of in people who remained ashore.

Conveyance of cholera on board ship.—It may be confidently stated that ships sailing from Indian ports suffer very little from cholera, as immunity of the P. & O. vessels from the disease sufficiently proves. When cholera does occur, Cuningham states it is limited to persons who have come from an infected locality. Pettenkofer[1] questions if cholera can occur on board ship unless in the persons of those imbibing the germs before embarkation. If, however, Cuningham's theory of atmospheric change or epidemic influence descending like irregular rains were correct, there would be no reason why it should not fall on ships. Pettenkofer's theory that the seed or germ of the disease must be carried into an endemic locality to spread is more in accordance with what he states of ships, than the theory of Cuningham is with his statement. The only countries on the globe in which cholera has not occurred are the islands of the South Pacific, Australasia, the Cape of Good Hope, the islands of the North Atlantic, and the western coast of South America. These countries are all separated from India by a wide expanse of ocean, and have little commercial intercourse direct with that country. But while there is positively no instance of cholera being conveyed to the Cape, or to Australasia (until last year when introduced into Brisbane by the 'Dorunda' after touching at Batavia), it is supposed to have been carried to the Mauritius from India in 1854 by coolies in the ship 'Sultan;' in 1856 by the 'Futteh Mahomed,' and in 1859 by the 'Topaz.' There are several instances on record of its having been conveyed by a recurrence of cases from India to the Red Sea ports, Egypt, and even to England, and there are still more examples of its

[1] *Lancet*, Dec., 1884.

conveyance from one Indian port to another. The fact is, if cholera is introduced into a ship it may spread ; but owing to fresh sea air, and the ordinarily good sanitary arrangements of ships in the present day, it rarely does so. If a ship is at sea with a clean bill of health longer than the period of incubation of cholera (*vide* p. 112), there will be no cholera imported by the passengers or crew, although it still may be conveyed by certain kinds of cargo (*vide* p. 107). Hence the absurdity of passenger quarantine in Egypt. Lastly, while there is no instance of cholera on ships previous to communication with an Eastern port, cholera usually appearing earlier at European ports than inland, indicates importation from the East.

The question of contagion.—The varying experience and opinions regarding the *contagious character* of the disease led to the idea that cholera may be contagious under certain circumstances, but not under certain other conditions, and therefore that it is not contagious under the ordinary acceptation of the term.

Cholera patients, it was asserted, could not communicate the infection to others unless by means of the discharges which they pass. Persons attending them run no risk of contracting the disease, provided they are protected from swallowing or inhaling the organic poison passed by the sick ; but in badly ventilated rooms, this organic matter having been disseminated in considerable quantities through the atmosphere, may be taken into the system by attendants, and so poison them. It was argued that importation does not necessarily imply contagion, and that it is impossible to apply to cholera the ordinary views about contagion. While some were of opinion that the poison, even if taken into the system, was only contagious under indefinite states of weather, atmosphere, season, or constitution ; others held that it is contagious only in its endemic area, and although the poison might be conveyed by man from the endemic

area to a non-endemic position, it then ceased to be contagious, and could not be again generated. Cases might occur from contact with imported seeds, but these would not result in an epidemic unless the local conditions were favourable, otherwise the place would escape in spite of the importation. Pettenkofer is the principal exponent of these theories, and calls cholera an *ectogenous* disease, as being the result of a micro-organism developing apart, but in the vicinity of an infected patient, in contradistinction to an *entogenous* disease where the virus is transmitted direct from the sick to the healthy. Pettenkofer's views have been adopted by several Indian officers. Bellew[1] states it cannot be carried from an area in which the disease is prevalent to another in which it has not made its appearance in the natural course of seasonable development. Cuningham says the endemic area is not well-defined, but shades off indefinitely. This endemic area has been limited to an absurdly small extent, as to part of a ship, the proof tended being the fact of cholera on board among troops not spreading to the crew. On the other hand, the endemic area has been extended to any part of India, as the disease spread there when it was regarded as infected. I do not believe in this endemic area. I do not think there is any place where cholera being introduced may not spread, although not by actual contact; or any place in temperate or tropical climates where it may not originate. That some localities and climates are more favourable to spread and origin than others is patent, and if the endemic area were limited to such localities I should not object to the term. But the endemic area, according to the inventors of the term, is wherever cholera does spread!

Incubation of cholera.—The weight of evidence tends to the belief that the incubation of cholera averages $3\frac{1}{2}$ days. But it may occur in a few hours, or be extended to 11 days. Pettenkofer gives $2\frac{1}{2}$ days to 5, with an average of 3. Sicar

[1] *History of Cholera in India from* 1862 *to* 1881.

gives 2 to 3 days, with an average of a week. Ziemssen says 5 to 7 days; Bryden, as the result of the investigation of 611 cases who remained but a few hours in infected places, 3 to 11 days. The International Sanitary Commission of 1874 were of opinion that incubation does not exceed a few days. Bain gives 40 hours. Waters says 4 days is an unusually lengthened period. Macnamara's cases (*vide* p. 94) all occurred within 36 hours. Other writers mention from 2 to 14 days. At the commencement of 1882, shortly after the Egyptian authorities first instituted quarantine against Bombay, I brought the subject before the Bombay Medical and Physical Society. At a full meeting, the members present being both Native and European practitioners, it was voted unanimously that the quarantine regulations against Bombay were useless and unnecessary. This conclusion was arrived at chiefly on the grounds of the incubation of cholera being limited to ten days, a period too short to admit of the passage of a vessel from Bombay to Suez. It would be impossible to assemble a body of gentlemen having more experience of cholera than those taking part in the discussion, and their deliberately expressed opinion was doubtless calculated to strengthen the position of the British authorities in requiring the removal of the vexatious Egyptian cholera quarantine, as also to settle the question of the incubation of cholera.

Briefly, the views of cholera as given at length in the foregoing remarks may be thus summarised:—

1. According to some, cholera depends on no definite entity, but is due to certain atmospheric and telluric conditions. When these conditions prevail cholera occurs, but the cases do not stand in causal relation. This theory does not admit cholera into the rank of infectious diseases.

2. Others maintain the character and course of every epidemic point clearly to cholera being dependent on human intercourse, every epidemic having its origin in importation

from a country where cholera had previously occurred. This theory places cholera in the rank of infectious disorders. But the upholders of this theory group themselves into two classes, viz., those who maintain cholera is contagious—that is, communicable from person to person by means of the excretions; and, secondly, those who do not admit of direct contagion, but regard changes in the evacuations, cast into a suitable medium as the cholera virus, which finds access to the human body through air, food, or water.

3. Others are of opinion that the evacuations of the cholera-stricken do not contain or form the virus, but regard it as a product or organism, altogether extraneous to the body.

4. Lastly, there is the ptomaine theory mentioned at p. 98.

The author's idea of cholera is as follows. There is an unknown atmospheric condition, occurring more frequently in eastern than in western countries, which, meeting with certain, but unknown, conditions of matter, presenting most usually in unsanitary localities, generates an invisible, impalpable, imponderable, chemically and microscopically unrecognisable germ or poison, which may be conveyed in all directions by human beings, by cholera excreta, by clothing, by some varieties of merchandise, by water, by food, by insects, especially flies, or through the atmosphere, to an undiscovered extent; probably in a more certain manner, in a more virulent form, and to a greater distance if favourable atmospheric influences exist. These ideas do not credit Bengal with being the birthplace and home of cholera. They are opposed to the assumption of cholera being a malady which must spread in a definite direction from east to west, and arrive at a certain place by a certain date, and they do not limit the mode of dissemination to one particular channel. On the contrary, they assert the *de novo* origin of cholera under favouring circumstances in any country. They are

based on the belief that the spread of cholera in a definite direction from east to west is a mistaken notion, arising from false reasoning on the fact of cholera epidemics being more frequent in the East. They are further based on the belief that western cholera epidemics would occur just the same if there were no eastern epidemics, and on the disbelief that the western epidemics referred to at p. 75 and regarded as the spread of eastern epidemics were consequent on the onward march of cholera. It is submitted that because cholera occurs in 1882 (or in any other year) in Bengal, the occurrence of cholera in Europe in 1885 (or in any other year) should not be regarded as a sequence to the Indian epidemic. It might, with equal reason, be advanced that the occurrence of so-called malarious fever in Europe is due to the onward march of tropical malarious fever, which in India is, like cholera, always most severe and prevalent. Or it might with equal reason be asserted that the occurrence of typhoid fever in India is due to the eastern progress of that malady from Europe. Had similar researches been made into cholera as it affects countries east of India, there would be similar reasons for asserting an eastern progress of the malady. But countries east of India are less known, and attention has not been directed so much to the East as to the West.

All this I submit is more in accordance with the general and local history of cholera than the theories of Bryden and Cuningham, which are promulgated from the general while ignoring the local history of the disease. There are instances of the outbreak of cholera where no rational explanation is admissible except that of *de novo* origin, while the communication of the malady by human intercourse, by clothing, by some varieties of merchandise, by water, by food, by insects or through the atmosphere, explains the origin of attacks which are otherwise unexplainable, and such explanations appear as certain as the exact sciences.

Mortality from Cholera.—Cholera, next to fever, is the most destructive of tropical maladies. In 1879, of 4,975,052 registered deaths in India, 270,553 were from cholera. On an average of bad years the mortality may be accepted as 100,000, in good years 10,000. The mortality of those attacked attains to three-fifths during the earlier part of an epidemic, and to two-fifths during the latter, or an average of 35 per cent. From one-fourth to one-fifth of the deaths occur during the stage of reaction.

Symptoms.—Cholera may commence suddenly or after malaise or painless diarrhœa, which may extend from one to ten days, or even longer. During this initial period of the malady, when remedies would probably be efficacious, sufferers often neglect to apply; the peculiar apathy mentioned as characterising the later stages of the disease seeming to be more or less present from the first. The seizure very often occurs during the night, especially to Europeans, while it has been noticed that natives often suffer soon after a full meal. Spasmodic griping in the bowels is felt, followed by vomiting and purging, first of the contents of the stomach and intestines, and then of large quantities of a turbid whitish, almost odourless fluid, with flocculent atoms floating in it, found under the microscope to be epithelium-casts thrown off by the mucous membrane of the bowels. The evacuations from the bowels of these 'rice water' stools may amount to fifteen or twenty in the course of a few hours, and at first they are discharged with great force, and are followed by a sense of relief, although by a peculiar feeling of exhaustion at the pit of the stomach. Vomiting may also be equally frequent, and the ease with which the cholera-stricken vomit is very remarkable, the material often passing up as if by a simple regurgitation. Simultaneously with the vomiting and purging, or very soon after, severe cramps come on, sometimes commencing in the fingers and toes, sometimes alternating with tingling, and rapidly extending to the calves, thighs,

and abdominal muscles. With all this there is at first scanty, high-coloured, often albuminous urine, deficient in urea, and then total suppression of urine; a burning sensation and feeling of constriction at the epigastrium which is sensitive to pressure; a white tremulous tongue, and a bitter taste in the mouth; great thirst, little or no saliva being secreted, and an urgent desire for cold drinks; a feeble pulse, but more frequent than normal, rising probably to 96; a cool skin and no fever, although the patient often complains of heat and oppression, and prefers to lie uncovered; noises in the ears may also be complained of; there is much restlessness—the patient often tossing about the bed; lastly, a rapidly progressing exhaustion is evident. The case now stands on the verge of collapse. Should this succeed the pulse becomes quicker but hardly perceptible, the discharges cease, and so do often the cramps. The skin is covered with cold perspiration, has a sickly smell, and a bluish tinge, very different from the pallor of hæmorrhage. The nails and lips, especially, assume this unnatural appearance. The whole body shrinks and seems to wither visibly, the genital organs are retracted, and the skin of the fingers is corrugated. The voice is husky and faint, and the tongue and expired air are cold. The intelligence is ordinarily clear, but there is a complete apathy as to the result. The countenance now assumes the aspect of death, the eyes are sunken and have a glassy appearance, the temperature in the armpit falls to 94°, although it may be much higher in the rectum; the pulse becomes imperceptible, there is hiccough, and stools may be passed unconsciously. Death, says Chevers, 'is marked by a peculiar physiognomy,' the body is of a bluish grey, with collapsed features, hollow cheeks, pointed tongue, deeply sunken and closed eyes, although the patient still possesses consciousness, and may be roused. Often two or three hours before death some return of heat in the scalp, forehead, or even over the chest, may present. This is to be

regarded as due to relaxation of the minute arterial branches, and is usually a fatal sign.

In those cases which recover the vomiting and purging gradually subside, the skin becomes warmer, the pulse fuller, the voice regains power, urine is again voided, colour appears in the stools, and the patient falls into a refreshing sleep. Even in apparently hopeless cases recovery may take place. So long as the patient has strength to vomit the case is not desperate. But we can never be satisfied that the danger of cholera is over till natural urine is secreted, and the average time of passing urine is seventy-two hours after seizure.

When the prostration has been great the reaction is apt to be violent. A fever of low type, with rapid pulse and heat of skin, and attended often with alarming cerebral symptoms, succeeds, while the urinary secretion may again become scanty. The cerebral symptoms referred to have been considered due to uræmic poisoning consequent on the non-elimination of urea by the kidneys. But this may be questioned, as head symptoms are often due to simple reaction after collapse, or to the excessive use of stimulants or opium, or in some instances to the existence of sugar or bile in the blood. In cases in which uræmia sets in, whether it is followed or not by fever of a low type, there is at first but little heat of skin with a slow pulse. The patient is wild and restless, eventually becoming drowsy; the kidneys act imperfectly, and the urine is greatly deficient in urea, and usually contains albumen. As collapse is the first great danger, so uræmia, or as Chevers prefers to call it, *cholo-uræmia* is the second, while simple reactionary fever without uræmic poisoning is the third.

Convalescence from any serious form of cholera is often slow, irritability of the intestinal canal remaining for weeks or months. In mild cases, however, the recovery may be complete and rapid.

Peculiar Symptoms.—Poynanski, quoted by Horton,[1] remarks on a slow pulse—as low as 45—for some days or weeks as pathognomonic of the approach of cholera. After the premonitory symptoms diarrhœa and malaise are absent. Or diarrhœa may be absent and malaise more pronounced, the person feeling very uncomfortable and tired. So frequently is premonitory diarrhœa absent that Surgeon-General Pringle[2] considers there is usually no such stage. In most great outbreaks at first persons die suddenly from collapse without distinctive symptoms, often without vomiting or purging, or after one or two loose stools. This was noticed in Bombay in 1883. More recently cases occurring in Poona are reported to have assumed the form of the 'sweating sickness of the 15th and 16th centuries.' In instances where death occurs from sudden collapse without vomiting or purging there may be violent cramps of the lower extremities. Clarke has recorded an outbreak of disease on the Coromande Coast in 1750, having all the symptoms of cholera *except* purging. Chevers mentions two epidemics in Bengal, having all the symptoms of cholera excepting the stools being tinged with blood. A similar outbreak has been described as *hæmorrhagic cholera* when the stools consisted of mucus or gelatinous fluid mixed with blood. Rice-water evacuations may be absent in the old and feeble, or rice-water may be found in the intestines after death, none having been passed during life. Nitric acid sometimes gives a brilliant ruby-coloured reaction with rice-water stools, resembling that often seen in nervous diseases, and showing the presence of small quantities of bile. Sometimes the stools are not turbid like rice-water, but colourless like blood-serum. While in some cases cramps are absent, cramps and twitchings have been noted without other symptoms (tetanic cholera, *vide* p. 73). Also sensations of 'pins and needles,' or anæsthesia, or paralysis, are occasionally present. Vomiting of worms has often been

[1] *Diseases of Tropical Climates.* [2] *Med. Times,* Sept., 1884.

noticed both during and after the attack. In the report of the cholera of Bombay in 1883, it is stated, 'Another strange feature is the vomiting of worms by those who have recovered from the primary symptoms;' but Bombay being, like many other parts of India, a very vermifuge locality, the explanation is easy (*vide* Worms). In the stage of reaction there has been noticed obstinate vomiting of thick greenish material, probably bile-pigment acted on by some acid. This may last for some days, and is most common in those of intemperate habits. During reaction there is sometimes a double heart-beat.[1] In one epidemic delirium occurred in 9 cases out of 110. Insane persons suffering, have sometimes recovered their senses temporarily. The connection between cholera and fever has already been referred to (p. 90), and it must not be forgotten that cholera in its commencement may simulate an attack of ague or terminate as fever, or there may be no fever. In the Bombay epidemic of 1883 febrile symptoms first showed, vomiting and purging then taking place. Dickson,[2] speaking from his experience in India, states an *increased* flow of urine, so often remarked in ague, is sometimes a symptom of epidemic cholera. Sometimes, instead of albumen, urine if passed contains sugar. Occasionally from cholera patients there is an extraordinary peculiar sickening smell likened by Murray to *arum maculatum*, or tainted fish. In females there is sometimes a sanguineous vaginal discharge when the person is not menstruating. The period and profundity of cholera is very variable, and the greater or less lividity of the countenance has given rise to such appellations as 'blue' and 'black' cholera. After death a remarkable contraction of voluntary muscles sometimes occurs, which has led to stories of persons being removed to the dead-house while yet alive. These spasmodic contractions are due to post-mortem relaxation of arteries and flow of blood. Occasionally persistence of heat

[1] Roche, *Lancet*, 1883, p. 139. [2] *The Unity of Disease.*

obtains for a longer time than ordinarily after death. Among the peculiar *sequelæ* of cholera are to be noted imperfect reaction, convulsions especially in children, nettle-rash or roseola choleraica, retention or suppression of urine, constipation, meningitis, gastric affections, enteritis, hæmorrhages from the bowels, diphtheria, bronchitis and pulmonary congestion, sloughing of the cornea, sloughing of the scrotum, abscesses in different parts of the body, the formation of coagula in the right side of the heart or pulmonary arteries. In females if the disease occurs during the early months of gestation abortion generally results, either at the period of attack or soon afterwards. At a later period of gestation the fœtus is generally killed, and premature expulsion follows.

Diagnosis.—There can be no difficulty in diagnosing a typical case of cholera, although it may sometimes be difficult to draw the line between cholera and diarrhœa, or to decide whether the malady is really cholera or fever, especially when diarrhœa takes the place of the cold fit of ague. Cholera, however, rarely begins with rigors, and fever is not ordinarily attended with cramps and rice-water stools. In an obscure case, the fact or otherwise of cholera being in the neighbourhood would tend towards deciding the question. The diagnosis between typical cholera and ordinary diarrhœa is undoubtedly sufficiently easy, but there is strong reason to believe that some forms of diarrhœa are the same disease as cholera, only differing in intensity. It has been reasoned that on the principle of evolution diarrhœa of pythogenic origin may assume more and more a specific nature until it becomes genuine cholera. Summer diarrhœa is induced, if not by heat, at least during the season of greatest heat; the initial stage of cholera cannot be diagnosed from any other diarrhœa; summer diarrhœa is often associated with cramps; the diarrhœa of children assumes the likeness of cholera to such an extent as to have obtained the name *cholera infantum*. In short, all the earlier symptoms of

cholera, excepting rice-water stools and suppression of urine, are met with in diarrhœa. Whenever cholera prevails diarrhœa is increasingly prevalent. The question therefore arises, at what stage does the sufferer from diarrhœa become the victim of cholera. To regard the case as cholera only when two prominent symptoms (suppression of urine and rice-water stools) occur is scarcely consistent with the known fact of an attack commencing as diarrhœa and ending as cholera. The term *choleraic diarrhœa* has been struck out of the official nomenclature, but there are numerous cases which can only be correctly described by such a medium term.

It must be recollected that various causes will produce symptoms very like cholera. These are principally arsenic-poisoning, foul water, putrid fruit, and especially putrid fish. When a case resembling cholera occurs in the absence of any epidemic, it will be well to inquire into the possibility of such causes of ailment. Judging from the similarity to cholera of the symptoms produced by tainted fish, it does not seem improbable that a poison closely allied to that of cholera is produced in the fish-ptomaine.

Prognosis is most unfavourable at the beginning of an epidemic, and more favourable towards the end. Youth, health, previous good circumstances, and good hygienic conditions are all in favour of the sick person. Childhood and old age are the reverse. There is little hope of recovery when there is organic disease of the lungs, heart, liver, or kidneys, or when the patient is diabetic. Fatal signs are mentioned at p. 117, and favourable at p. 118.

Post-mortem appearances differ materially, *first* with reference to the stage of the disease at which death takes place; and *secondly*, with reference to the period which elapses after death before the body is examined.

External appearance.—Face and body are shrunken, and present a bluish pallor very different from the bleaching observed in those dying from hæmorrhage. There is loss of

weight, as much as thirty pounds having been noticed. A rise of temperature of the surface of the trunk occurs, often observable for some hours after death, due to arterial relaxation and flow of blood (*vide* p. 117). Muscular rigidity is often remarkable, as also are muscular contractions (p. 120). It is recorded of the Indian cholera of 1832 that the soldiers bound the limbs of their dead comrades, in order to dispel the idea of persons being removed to the dead-house while alive. These muscular twitchings are doubtless due to the same cause as the rise of temperature.

The digestive organs and viscera, in which explanatory changes might be expected, do not afford much information. The stomach has been found distended by watery fluid after early death, but is usually empty and contracted. Congestion of the mucous membrane of the stomach is found, especially after severe vomiting, and more decidedly in persons of intemperate habits, or when quantities of stimulants have been given. The peritoneal covering of the small intestines is often of a pink hue. The most constant and marked change is more or less redness of the lower part of the small intestines, or an 'arborescent venous congestion.' There is also destruction of epithelium, mostly through some two feet of the ileum immediately above the cæcum (which Macnamara regards as post-mortem), and enlargement of the glands. Sometimes the intestines contain rice-water fluid, at others semi-opaque mucous matter containing cylindrical epithelial cells. Occasionally there is a colourless gelatinous exudation with few or no cells. The comma bacillus is also usually found. The kidneys and liver are generally found congested, and the gall-bladder may be moderately full or distended. The condition of the spleen is very variable, it may be found distended, but reduction of size and weight has been noted.[1]

As regards the *circulatory system*, the periodical fluid has

[1] *Report of the Sanitary Commissioner with Government of India*, 1870.

been found scanty and slightly acid half-an-hour after death.[1] The pericardium and cardiac lining membrane of the heart is sometimes congested or even ecchymosed. Shortly after death the left ventricle is contracted, at a later period both ventricles are contracted and moderately full; but Macnamara found both sides full immediately after death. After death during collapse there is engorgement of the right heart and pulmonary arteries, the left side and pulmonary veins being nearly empty. But light or dark clots may be found on either side of the heart, their presence appearing to depend more on the length of time elapsing before examination than on the disease. After death, during the algide stage, the lungs are usually extremely contracted.

As regards the *blood*, chemistry has not shown increase of urea, as might be expected. An apparent increase in the number of both white and red corpuscles has been noticed, probably due to thickened blood, and the red discs have been observed flattened and pale. Small elongated bodies resembling those of acetic fermentation have been found between the corpuscles, accompanied by slight acidity of the serum (French Egyptian Mission of 1883). But the state of the blood varies with the stage of the disease. It is always, however, more dense and thicker than natural, becoming lighter on exposure to air. No changes have been observed in the brain or spinal cord, except a general paleness and scantiness of fluid in the cavities of these organs.

Pathology.—Admitting the existence of a poison as the cause of cholera, two theories have been proposed. First, the poison entering the blood acts as an irritant affecting the nerves, and through them the coats of the smaller arteries of the lungs, obstructing the circulation of fluid through those organs, and thus the blood receives less oxygen than in health; the deficiency of oxygen in the circulating fluid leading to the symptoms of the algide stage. The second

[1] *French Scientific Egyptian Mission*, 1883.

theory maintains that in consequence of the alvine flux the blood loses its serum, the corpuscles their water of composition, and becoming dehydrated they can no longer fulfil their office as carriers of oxygen, and hence algide symptoms are induced. These theories merge closely, inasmuch as they both recognise the want of oxygen in the circulatory fluid as the chief factor in the production of the collapse of cholera. Briefly stated, the pathology most generally accepted is obstruction to the pulmonary circulation from the right to the left heart consequent on non-oxygenation of the blood and the tissues, and exudation of the watery parts of the blood into the intestines. The intestinal functions are reversed—instead of absorption taking place an exactly opposite condition is progressing; an exasmose into the bowels of the serum of the blood. Endosmosis ceasing, all vital operations cease also; and the result is asphyxia. Suppression of urine was attributed by Dr. G. Johnson to the fact of the blood having lost so much of its watery portion through the bowels as to render further secretion impossible. It is, however, very evident that if in consequence of failing circulation no blood reaches the kidneys or liver, they cannot elaborate their secretions.

Not admitting the existence of any specific contagion or morbid infecting agency, cholera has been ascribed to the results of simultaneous and abnormal superabundance of blood in, and preternatural activity of, both the spinal cord and sympathetic nervous centres, constricting the arteries, impeding the pulmonary circulation first and the general circulation afterwards, preventing the flow of blood necessary for secretion, and eventually shutting off the flow to the surface of the body.

It is by the action of cerebro-spinal nerves that voluntary muscles are brought into motion, that the various glands throughout the body are made to secrete, and that the manifold impulses made on the peripheral ends of sensory

nerves are conveyed to nervous centres. But Bernard found that the involuntary muscles constituting the muscular coat of arteries are as thoroughly dominated by nerve-force as voluntary muscles, and that such nerve-force emanates from the sympathetic. Owing to the power of the sympathetic of contracting arteries, Bernard named it the frigorific nerve. Hence is based Chapman's theory, as above noted, of cholera being due to preternatural activity of the nervous centres.

Treatment.—When we remember the numerous and diverse remedies proposed for other diseases, it is scarcely matter of wonder that, in a malady so rapid and dangerous as cholera, where so little light is derived from pathology, multitudinous and totally different treatments are recommended. It would indeed be difficult to name a drug which has not at one time or other been proposed for cholera. Those who considered the disease due to malaria have advocated quinine. Those who regard it as a nervous affection have used chloroform or ether, or spinal ice-bags. Some have withheld water, others have made their patients drink water to the *finale*.

Gull used the wet sheet. Dr. Parkes, with greater theoretical probabilities of success, used the warm bath. Some have placed faith in sulphuric, some in nitrous or nitric acid. Regnole, of Rome, and Hayen, of Paris, have faith in the *æthiops mineralis*, which is said to be destructive to the common bacillus. Dr. Graves brought forward sugar of lead and opium as a never-failing remedy. Raspail, in Paris, asserted camphor to be an infallible cure. From an idea that the salts of the blood were deficient arose the plan of saline enemata, and venous injections were recently used in Naples as a new treatment. Bleeding and calomel have, of course, had their full trial. The latest advocate of bleeding was Deputy Surgeon-General Playfair, who in 1861, reviving the discarded practice of Annesley and Twining, bled his patients, although to a much smaller

amount, taking only two or three ounces of blood away at the commencement of the stage of collapse, and at the same time giving a stimulant. This, he stated, while relieving the heart increased the force of the circulation, and he reported greater success from this treatment than from any other, although admitting uncertainty as to its being be equally successful in every type of cholera. Many have used calomel; in 1848 large doses were given to compel the liver to act, subsequently smaller doses to solicit the liver to act. Neither have croton oil and ox-gall, castor oil and sulphate of magnesia, with other drastic and mild cathartics, been forgotten. Chloral, internally, has of course been used, and the hypodermic injection of chloral into the muscles has been reported to allay spasm.[1] Reddie recommends one grain to ten minims of water as the strength of the injection. The hypodermic injection of morphia over the stomach has been stated to allay both sickness and spasms.[2] Hypodermic injections of liquor ammoniæ have been reported on favourably by Young,[3] and the reverse by Christison.[4] Injections of solution of chloride of mercury have also been spoken of favourably.[5] Sutherland[6] tried injections of chlorate of potash without much success. Other saline injections have also been used. Stewart[7] commends a combination of spirits of nitre, spirits of chloroform, and tincture of catechu, taken internally. Francis[8] stated any one using the tincture of cantharides will be gratified by the result. Transfusion has also been tried, Dr. D. Smith giving his blood *ad deliquium* without good result to the patient. Plugging the rectum has also been seriously proposed, if not practised. Dr. Macdowal,[9] Bombay, recollecting that Dr. Richardson,[10] in 1854, established the fact that

[1] Hamilton, *Lancet*, 1883, p. 973.
[2] *Lancet*, Sept. 29, 1883.
[3] *Ind. Med. Gaz.*, 1869.
[4] *Ibid.*, 1869.
[5] Reddie, *Ind. Med. Gaz.*, 1880.
[6] *Ind. An. Med. Sci.*, vol. x.
[7] *Lancet*, Sept., 1883.
[8] *Ind. Med. Gaz*, 1869.
[9] *Ibid.*, Oct. 13, 1883.
[10] *On the Coagulation of the Blood.*

water to a fifth of the weight of the animal may be injected into the peritoneal cavity, proposed such means for cholera. In one case peritoneal injection was performed years ago, and the patient recovered; but failure resulted in a second instance. Macdowal [1] also proposed injection of the cellular tissue, which, however, was also tried by Richardson[2] with even less favourable result. The same authority proved that water injected into the empty bladder is absorbed, and 'medication *ad vesicam*' has been advised by both Richardson [3] and Purvis.[4] The inhalation of oxygen gas, brought forward as a new idea at Marseilles in 1884, but which I tried in 1849, had no beneficial effects, and the same may be remarked of oxygenated drinks recommended by Dr. Lownds [5] in 1860. Jefferys suggested the diminution of atmospheric pressure over a large surface of the body by means of an air-pump, which was tried ineffectually by a French physician. Chapman [6] vaunts the spinal ice-bag or the *neuro-dynamic* treatment of cholera, on the theory mentioned at pp. 125–126 of cholera depending on congestion of the nervous centres, &c. The ice-bag is to be applied from the upper part of the cervical to the middle of the lumbar vertebræ, and he states, 'Precisely those persons who suffer most from bodily cold are most needing treatment by ice along the spine.'

Mustard and water emetics have been extensively used by Forlong, and various other emetics by different practitioners. Harkin, in the *Lancet*, August, 1884, gravely proposed the use of an epispastic solution behind the ears, which it is stated stimulates the pneumogastric. Some years back the height of credulity was required by a German in Calcutta, who announced the cure of cholera by the subcutaneous injection of infusion of quassia. Yet the latter,

[1] *Lancet*, Oct. 13, 1883.
[2] *On the Coagulation of the Blood.*
[3] *Medical Times*, Aug., 1883.
[4] *Lancet*, Oct., 1883.
[5] *Trans. Med. Phy. Soc.*, Bombay.
[6] *Cholera Curable*, 1885.

equally with many of the former, refers to what he considers a most successful practice. The fact is, however, as stated by Chevers, that the treatment which appears to be beneficial in one epidemic is generally found useless in another. The declaration of Elliotson, who tried mesmerism, still holds good, viz., that 'We are, unfortunately, ignorant of any decidedly beneficial specific treatment.' No drug up to the present time has been found of the slightest efficacy in averting a fatal issue. Hence Dr. Blanc and other experienced observers now assert that, so far as drugging is concerned, almost total non-interference is best; and he will best treat the malady who, guided by the symptoms in individual cases, so combats the tendency to death. Successful treatment of cholera depends much on the sanitary surroundings of the patient, on the care that is taken in nursing and supporting him, not interfering with him too much, but carefully watching and treating the symptoms as they arise.

In this way much may be done, if it only results in securing the confidence of the patient that he is not being neglected. In the first place perfect ventilation must be insisted upon, and from the very commencement the recumbent posture must be enforced, an india-rubber sheet being placed over the bed.

When cholera is prevailing, the slightest approach to diarrhœa should be at once attended to, otherwise it will probably run on to cholera. And most physicians who have seen much of the disease have for this premonitory stage their favourite remedies. Really it does not much matter what is given, provided the diarrhœa is treated in a rational manner. Chevers prefers tannic acid, ten grains after the first motion and five grains after every other. Playfair advised twenty drops of laudanum and three grains of cayenne pepper in half a tumbler of hot water. Macnamara when practising in the area of cholera was in the habit of

carrying with him pills containing one grain of opium and four of acetate of lead, so that no time might be lost in giving one dissolved in water. I would give thirty drops of chlorodyne in half-a-wineglassful of brandy (or more for those accustomed to spirits) in as much water, which are remedies usually to be found when wanted; or, if available and especially if sickness accompanied the diarrhœa, one drachm of chloroform, one drachm of aromatic spirit of ammonia, two drachms of chlorodyne, one ounce of brandy, a tea-spoonful for a dose in five or six ounces of soda water. If neither of the above are at hand I would give, if available, twenty drops of spirits of camphor every half-hour. If purging continues after two or three doses of the first-mentioned remedies, or after five or six of the camphor, ten grains of Dover's powder every three hours. To maintain the flow of urine one drachm of sweet spirits of nitre in two ounces of water every three hours, but not at the same time as the Dover's powder. As soon as possible if vomiting occurs, a mustard poultice should be applied to the epigastrium, and as the incidence of cholera is on the lower part of the small intestines the right iliac fossa should be counter-irritated. In any case the patient should be kept from the first in the recumbent posture and as quiet as possible, and until purging has stopped all solid food should be avoided, although tea, milk, arrowroot or sago, mutton or chicken broth, with a little good port wine, may be allowed if inclination for such things exist. Thirst may be quenched by plain cold water, and ice if available should be kept constantly in the mouth. Macnamara states the patient should be prevented from taking any fluid but what he gets from ice. Others have allowed water in moderation, some *ad libitum*. Neither do I think any harm arises from drinking water, but as a matter of fact most patients if they have ice crave little for water. Should cramps occur, the pain may be relieved by friction with the hand, or by cloths saturated with warm turpentine, or the

patient may be allowed to inhale some ether. Hot water bottles should also be applied to the feet. If purging and vomiting continue, the patient becomes as described in the latter stages of the disease, falling into a state of collapse. The period for any medicine, as chlorodyne, opium, or astringents, has now passed away, but every effort must be made to support the system and to maintain animal heat. In collapse it is useless giving medicines, as the functions of the whole intestinal tract are reversed. Instead of absorption taking place, an exactly opposite action, or endosmose into the bowels of the serum of the blood, is in progress. Therefore the stomach cannot absorb medicines, and they may accumulate, becoming the cause of much mischief by aggravating reactionary fever. If remedies are given at this stage and the patient vomits, he probably gets rid of them, if not, they do no harm at the stage when the organs should be at rest. The great desideratum in collapse is to keep up animal heat in every way which will not fatigue the patient. One handy and serviceable method of maintaining animal heat is by extemporising a hot air bath by means of a cradle above and a pan of hot ashes below a canework cot, on which the patient lies. Above all he must be kept quiet, and not be allowed to assume an erect posture. If the breathing is difficult, a mustard poultice may be applied to the chest. Thirst may be checked by a tablespoonful of brandy or three or four of champagne in a tumbler of water. No other stimulants should be given, but broth or raw meat soup may be offered frequently, but should not be pressed on the patient. Raw meat soup in small quantities, as a dessert-spoonful every ten minutes, will often be retained when everything else is vomited. The sudden cessation of vomiting when collapse is present must always be regarded as an unfavourable sign. The pulse may not be perceptible at the wrist, the surface may be cold, and the voice reduced to a whisper, but if the

patient has strength to vomit the case is not hopeless. Similarly, so long as urine is passed the case should not be regarded as hopeless, and with the view of encouraging this secretion mustard poultices may be applied over the loins. Although this measure and giving spirits of sweet nitre are recommended (p. 130), opinions differ much regarding the propriety of giving diuretics. Chevers is decidedly against their employment, as they must tend to irritate an already congested kidney, and Goodeve stated 'water is the best diuretic.' On the other hand, Francis strongly advocated the employment of cantharides tincture. I would limit the employment of diuretics to sweet spirits of nitre already recommended, which is the least irritating of diuretics, and also diaphoretic. When urine is suppressed, passage of a catheter is often useful, for although no urine is drawn off it is frequently passed afterwards. When the secretion of urine commences, the bladder should be watched, lest, as may happen, it becomes distended from loss of power to micturate.

The treatment of the reactionary fever of cholera must be guided by general principles. No two cases probably require exactly the same treatment. Too much should not be done, as probably injury has resulted from erroneous attempts to keep up the strength of the patient. Iced milk, or arrowroot, or raw beef tea, are all that should be allowed by the mouth, but nutrient enemata may be given every few hours. Patients do not usually sink in this condition from exhaustion, and over-feeding will bring back irritability of stomach.

Prophylaxis.—Among the numerous articles which have been advised as preventive of cholera are quinine, which Chevers thinks may have some prophylactic influence; salt, which has been strongly recommended by Beaman; dilute sulphuric acid, of which a drachm per diem in water has been advised; powdered hyposulphite of soda to be

taken with salt at meals; pepsine; black sulphuret of mercury, by which, Dr. Murino holds, we can make ourselves cholera-proof. In 1883, in Sweden, small copper discs worn over the stomach were found useless for cholera, but liable to create sores from verdigris; camphor-bags. Of these during a cholera season I advise a small daily dose of quinine, and plenty of salt with the food.

During epidemics of cholera it is especially advisable to keep the abdomen and feet warm, also to avoid chill or any extraordinary fatigue. The food should be moderate in amount, sufficiently nutritious and easily digestible. Anything which is highly fermentable should be avoided, but no sudden or radical change should be made, as it takes time to habituate to change of diet. What suits a person best at other times will generally do so during a cholera season. Purgative medicines should rarely be given when cholera prevails, and never at night. The first manifestation of disease should never be ignored.

In Indian practice for distribution to natives, or from dispensaries, I have reason to be satisfied with the cholera pill proposed by Surgeon-General Murray, and containing opium one part, assafœtida two parts, and black pepper three parts, made into a five-grain pill, to be taken every two or three hours in the initiatory stage. Dr. Murray does not regard this mixture as antidotal to the person but as stimulant to the stomach, which is thereby enabled to destroy the microbe [1] or poison.

On the expectation of cholera the following are the principal points:—great attention to the general sanitary condition, avoidance of over-crowding, attention to ventilation, examination of traps, sinks and drains, to see they are in working order, attention to the water supply. Among soldiers, diminution of night duties. Morning, tea or coffee; flannel belts.

[1] *Medical Times*, Aug., 1884.

When cholera occurs, the room and if possible the house should be vacated, cleansed and fumigated by chlorine gas, or nitrous or sulphurous acid gas. Evacuations passed should be received into vessels containing chloride of lime, sulphate of iron, or carbolic acid solution. Pettenkofer asserts disinfection of excrements does not diminish the spread of cholera, but still advises disinfection as conducive to cleanliness, the process having therefore advantages quite independent of the question whether the evacuations contain the cholera poison. Chevers holds similar views, and is in accord with the system of burning dirty linen and burying fæces, not because they are infectious, but because they are nasty. It certainly has not been proved without doubt that the fæces contain the poison, but it is possible, or even probable, that they do, and under this possibility their disinfection should not be neglected.

A prophylactic measure, much recommended after successful experience at Indian fairs, by Dr. Tuson, is burning sulphur. Fires about thirty yards apart should be kept burning for forty-eight hours, sulphur being constantly thrown on the fire. All the fires should be lighted at once, and about 4 lbs. of sulphur will be required for each fire during the period. The basis of Dr. Tuson's explanation of their efficacy is the germ theory.

Reports of the apparent cessation of cholera after volley-firing, morning and evening, have been made. It has been theorised that the concussion in some way altered the state of the atmosphere, or that the sulphur fumes discharged had some beneficial result. The cessation of cholera after such procedure was more likely *post* than *propter*.

When cholera attacks troops, removal from the affected locality (as long since practised by aboriginal Indian tribes, such as Bhils) and first proposed by Surgeon-General Murray for troops, has been found by experience to be the best means of averting the disease.

The following is an abstract of the most recent rules regarding cholera in India, promulgated to the Military Department:—

The necessity for constant attention to the sanitary condition of the locality is demonstrated by the fact of the outbreak of cholera being often sudden. If an outbreak appears probable, increased vigilance is demanded, but when the disease has actually appeared, more harm than good is likely to arise from attempting to cleanse foul drains or to remove nuisances. Especial care should be taken to prevent crowding in barracks and hospitals. The early treatment of premonitory symptoms is of the greatest importance, an appropriate medicines should be at hand. If cholera appears in the neighbourhood, soldiers and other residents should be warned against visiting the affected locality. Camping grounds, to be used in the event of an outbreak, have now been selected for all cantonments, and officers should make themselves acquainted with such localities.

On a case of cholera occurring in any building occupied by European troops, the room or portion of the building in which it occurred should be vacated. Those vacating should be isolated as much as possible, either in separate buildings or under canvas. Similar rules are applicable to families. The room vacated must be fumigated, and is not to be re-occupied within ten days.

If a second case of cholera appears among this particular body, they should be again moved. If a third case occurs within a week from the occurrence of the first, then the party should be immediately moved from the station to the camp. When separate detachments are thus moved into camp, it is advisable they should be kept distinct as far as possible. If the disease continue in such detachments, short marches should be made at right angles to the prevailing wind. Troops should not return to camp until they have been free from cholera for ten days.

As regards hospitals, if no separate building can be set apart, tents or suitable grass huts should be provided.

Special attention should be paid to everything tending to improvement of the general health. Every effort should be made to relieve soldiers from duties causing exposure and fatigue, to ensure that their food is wholesome and their clothing appropriate, and to promote every means of healthy amusement and occupation.

Inoculation for cholera has been recently much recommended by a Spanish physican, Ferran. It is remarked at p. 85, that when epidemics of cholera occur, exemption rather than seizure is the rule which may account for much of the

fancied success of this treatment, and it is remarked at p. 83 that one attack of cholera affords no exemption from future attacks. It is therefore difficult to understand how inoculation can do so. Inoculation for cholera has been proved untrustworthy.

Quarantine.—An author of repute has stated that the free communication from place to place confers such advantages on the human race, that we should not deprive ourselves of them even to protect ourselves from cholera. Unfortunately we cannot secure immunity from cholera by any system of quarantine, otherwise the above expression of opinion might be questioned. Gordon[1] shows that places under quarantine have suffered when other localities not under quarantine escaped. At Odessa a sentinel on quarantine took cholera, which, whether the man had or had not communication with the infected, shows the imperfection of quarantine arrangements. Many instances occurring during the European cholera of 1884–5 might be quoted, both in proof of the latter observation, and also showing that cholera does not invariably follow the panic flight of those from an infected locality. If, as I believe, cholera may be communicated through the atmosphere and be originated, as it usually is, *de novo*; or if, as others believe, there is no specific poison, but that it depends on atmospheric conditions, or on a ptomaine poison, the impossibility of maintaining freedom by quarantine is equally apparent. Quarantine can only be supported on the view of cholera being always communicated by human intercourse; facts showing that it is not even most frequently so communicated. Sir Joseph Fayrer truly remarked we might as well attempt to keep out a flight of locusts by a five-barred gate as cholera by quarantine. It is many years since general quarantine was enforced at the ports of the United Kingdom, and there is not the remotest chance of its ever being enforced again.

[1] *Notes on the Hygiene of Cholera.*

Ships will never again be sent to ride quarantine at the Mother-bank unless they are known to be attacked by yellow fever, or have cholera on board. It is the business of the officers of the Customs to inquire whether an arrival has communicable sickness on board; to detain the ship if there be such sickness, and to inform the port Sanitary Authority without delay. No ship is detained unless there is some reasonable ground of suspicion against her; the detention is only until such time as a leisurely and thorough medical inspection shall have been made; those who are found healthy are allowed to go; the sick are isolated, and the ship disinfected. This, with the precautions mentioned at p. 107, is the modern practice of inspection and isolation, based upon a better knowledge of the ways in which cholera spreads and upon our well-founded confidence in being able to deal with it. This practice stood the severe strain put upon it by the Baltic and North Sea commerce in 1873; it proved efficacious in 1884, on the arrival of the troop-ship 'Crocodile,' and we may take it that its superiority to quarantine, which is the arbitrary detention of all and sundry, has been demonstrated conclusively. I therefore again declaim against such absurdities as quarantine by European ports against vessels from India with a clean bill of health during the voyage, which must occupy more than the period of the incubation of cholera (*vide* p. 112).

Merchandise arriving, as it must often do, from localities in which defects of sanitation exist, exposed as it frequently is to direct contact with the epidemically diseased, cannot be free from the suspicion of carrying with it the *materies morbi* of disease, and of thus becoming a new centre of infection when the climate or predisposed constitutions of those among whom it may be placed, favours the development of the poison. The conveyance of cholera by merchandise is a risk which must be incurred. For if cholera-poison may remain dormant in some kinds of merchandise as it appears

to have done (*vide* p. 107) during fourteen or fifteen days, there is little reason to doubt that it may so remain for an indefinite period. But the case of the 'Columbian' (*vide* p. 107) exemplifies very forcibly the meagre protection which can be afforded by quarantine. For even if the 'Columbian' goods had been taken on shore for the purpose of depuration, such procedure would not have prevented the mischief which apparently arose from contact to those unloading. An instance to the point is that of the French transport 'Sarthe' which passed through the Suez canal *en route* from China to Toulon in April 1884, her arrival at the French port being followed by an epidemic of cholera originating among the persons on board. As the 'Sarthe' had no cholera on board since April 5, and yet could excite an epidemic when she was being unloaded and cleared out at her port of arrival in the third week of June, it may be accepted that a week or two's detention at Suez would have made no practical difference. Neither would any system of fumigation of passengers or merchandise make any practical difference. We cannot fumigate the interior of the human frame, while fumigation would in some instances be too destructive to merchandise, and in all cases too uncertain to be worthy of serious consideration. At p. 80 it is noted that the incidence of cholera is most severe in unsanitary localities, and at p. 114 it is shown that cholera may, and usually does, originate *de novo*. The inference is that we must trust to good sanitation rather than quarantine for immunity from cholera. It is on local precautions and not upon quarantine that a community must rely for protection against disease. Fortunately this is recognised in England and India. Experience has shown that our substitute for quarantine gives the maximum of freedom with the minimum of risk. But on the Continent quarantine is still regarded as the panacea, and sanitation is neglected.

CHAPTER VII.

CHYLURIA.

Synonym: Galacturia; chylous urine.

ALTHOUGH cases have been noted in Europe, chyluria may be defined as a disordered condition occurring chiefly in tropical and semi-tropical countries, manifesting itself by a milky appearance of the urine, accompanied usually by more or less distinct traces of blood-deterioration. Often there are no premonitory symptoms, but it is generally preceded by mental depression, debility, and pain in the loins, bladder, or perinæum, which is suddenly followed by a discharge of milky urine. It has been noticed at all ages, in both sexes, and in all conditions of life, but most frequently presents in the anæmic. It sometimes endures for years without apparent ill results, but those affected have been known to die suddenly —perhaps from embolism—without any other recognisable disorder. Occasionally there is distinct hæmaturia, which may alternate with the chylous condition of urine. Sometimes serous discharges take place from the lymphatics of various parts of the body, usually the axillæ, groins, or scrotum. Such cases have been termed *lymphorrhagia*, or the *lymphatic diathesis*, and may be independent of, or alternate with chyluria. Often they occur as the forerunner of, or in apparent connection with elephantiasis, and have been designated *elephantiasis lymphangiectodes*, *nævoid elephantiasis*, and *varix lymphaticus*. But although these affections may co-exist, chyluria is sufficiently often observed

as a separate malady without scrotal or other affection to warrant its description as a distinct disease. The occasional co-existence of these maladies, and the fact that Lewis, in 1870, found numerous hæmatoid worms (filaria) in specimens of milky urine, and in 1872 found the *filaria sanguinis hominis* in the blood of man and in persons known to suffer from chyluria, have given rise to the idea that the different forms of chyluria and elephantiasis are originated by these filaria, which in the causation of chyluria act injuriously by giving rise to rupture of the delicate lymphatic channels in which they circulate, thus allowing escape of nutrient fluids. Carter, however, advocated the view that a distinct connection exists between the chyle-carrying vessels and the urinary tracts, thus allowing a leakage from the former into the latter. Bernard and Robin opined that the condition of urine is a symptom of piarrhæmia or fatty blood, probably due to digestive derangements, or to liver derangement produced by filaria in that organ. Roberts surmised a sort of eczema of the bladder caused by a fatty condition of blood. But excepting the existence of hæmatoid worms and an anæmic condition of the blood, no alteration in this fluid is apparent, while post-mortem examination has not demonstrated the existence of any connection between the urinary tracts and chyle-carrying vessels, nor of eczema of the bladder. In fact, post-mortem examination has thrown no light on the malady, neither renal nor bladder disease being detected.

The malady known as *hæmaturia Braziliensis*, and also the Egyptian hæmaturia, have been associated with an entozoon, and have been surmised to be the same disease. But from the recorded character of the worms found in Brazil and Egypt it seems probable they are distinct varieties from that discovered in India; the difference, as Lewis surmises, eventually, perhaps, offering a satisfactory explanation of the

discrepancies observed in the character of the urinary disorders in the different countries.

Chylous urine emits a milky odour, and after standing coagulates, the clot decomposing in a few hours. In some instances the clot is pink from admixture with blood. Under the microscope, while lymphoid and red corpuscles are seen, tubular casts are rare. The specific gravity varies from 1007° to 1020°. Shaken with ether the urine loses its milky aspect. Nitric acid and heat throw down a precipitate. These facts, and that of the coagulability of the fluid, indicate, as Lewis points out, the presence of fat, albumen, and fibrine, all abnormal constituents. The amount of such abnormal constituents varies in different specimens, possibly in accordance with the quality of food taken.

Although næmatoid worms have been found in the blood, in the urine, and in the serous discharges, when they occur, of persons with chyluria, worms of similar description are found more frequently in the blood of those not suffering from chyluria. We cannot, therefore, yet regard them as the cause, although it may be admitted as probable; as also their introduction into the blood by mosquitos (Chap. XV.).

The *treatment* is unsatisfactory. Good nourishing diet, containing a large proportion of vegetable material, with anti-scorbutics, and gallic acid in half-drachm doses thrice daily, has seemed the most beneficial. Discomfort of the patient is somewhat relieved by a tight belt round the loins. The malady sometimes disappears without any remedy, and may persist in spite of all endeavours.

CHAPTER VIII.

CONSTIPATION.

Among the various causes on which constipation depends in India the most common are: 1. Some liver derangement, such as torpidity or chronic inflammation (*vide* Liver Diseases). 2. Deficit action of the small intestines. 3. Torpor of the large intestines. 4. Acute or chronic typhlitis.

When constipation results from deficient action of the small intestines, there is no excessive accumulation of fæcal matter, but a sluggish action, the stools being moderate in amount, very dry, and generally, but not always, light in colour, indicating accompanying torpidity of the liver. In most cases there is an uncomfortable feeling of dull pain at the back of the head, while the tongue looks small, and is a little red at the tip and edges. The mouth contains viscid mucus, indicating the condition of deficient secretion prevailing in the bowels. There are also loss of spirits and of appetite, with probably more or less flatulence, and slight colicky pains. This form of constipation appears often to be a result of so-called malaria, prevailing as it most commonly does in unhealthy malarious districts. Purgatives in such cases are not much required. What is wanted is the presence in the intestines of more liquid. A glass of cold water taken every morning on rising is often beneficial; or, this not succeeding, two or three drachms of sulphate of soda and two grains of quinine should be dissolved in a pint of water and taken as a morning draught, while a quarter-grain

of *ipecac*, and a half-grain of *extract of nux vomica* may be taken thrice a day. These remedies, aided by such means as fruit early in the morning, oatmeal porridge for breakfast, smoking after breakfast, brown bread eaten instead of white, the avoidance of pastry, regular exercise, and regular but not hurried visits to the w.c. will generally be successful. Such measures may be assisted at first by Hunyadi Janos waters.

When the large intestines are in fault, the tongue is furred, the breath fœtid, the complexion sallow. There are occasional attacks of colicky pain, the stools are hard, dark, or mottled—often hard at first, but afterwards semi-liquid, at other times small scybalous masses only are passed, with a considerable quantity of gas; or the ejecta may consist only of mucus, gelatiniform in appearance, or of long worm-like masses, resembling entozoa. These discharges are in some cases unusually offensive in their character, and thus induce increased distress to the patient. In rarer instances the evacuations present a frothy, yeast-like appearance, as if the fæcal contents of the colon had undergone fermentation. Another symptom, and a very characteristic one, is flatulent distension of the colon; this distension comes on several hours after a meal, and is often a source of annoyance to the patient during the night. Hæmorrhoids often complicate the condition, especially when from neglect accumulation is suffered to remain in the rectum. The general strength at first is not much impaired, but there is an inability to bear wonted fatigue. In other cases there is general depression, almost amounting to melancholia, inability to take active exercise, or to grapple with the worry and fatigue of ordinary life. In men the bladder often sympathises, and there is sometimes frequent desire to pass urine. In women I have found this state of the colon associated with dysmenorrhœa and ovarian irritation. If accumulations are allowed to remain constipation may alternate with diarrhœa, the evacua-

tions containing much bilious mucus; or a dysenteric condition may be induced, for the retained secretions undergoing decomposition irritate the mucus membranes on which they lodge both mechanically and otherwise, and thus may originate ulceration. This form of constipation is more likely to occur in India than in a temperate climate, the bowels, especially the colon, partaking of the general debility and want of tone resulting from long residence in the tropics, and becoming unable to expel their contents. Or in some instances cicatrices or contractions arising from former dysentery may add to the obstruction, when a want of tone is superadded at a later period of life. It may also be held in mind that accumulation in the larger bowels may be an obscure symptom of approaching cerebral softening, and this is especially to be feared if the person has previously suffered from sunstroke.

It is well to note that some individuals suffering from torpor of the large intestines state their bowels are regular, simply because they visit the closet daily, when in reality they suffer from constipation, as they only pass small hard fæcal lumps. Occasionally there is straining with hard lumps and watery discharge, the result of the irritation they cause. This is mistaken for diarrhœa, instead of being recognised as the effect of constipation.

The class of persons mostly coming under treatment for this form of constipation are old residents in India whose constitutions are impaired, and who are more or less cachectic, and powerful medicines are not advisable. Occasional doses of castor oil, or sulphate of soda, sometimes one sometimes the other acting best, are suitable. A grain of ipecac taken early in the morning is often useful. But the colon is so far removed from the stomach by the whole length of the small intestines, that greater benefit results from other measures than from medicines given by the mouth. Rubbing or kneading the bowels gently will sometimes promote

healthy action. But the periodical use of warm water enemata will prove the better course; or, if flatulence is a prominent symptom, the injection of powdered charcoal in thin, warm gruel will often afford considerable relief. In most cases the contents of the bowels require to be softened, and therefore the use of warm enemata is particularly appropriate. The first indication of cure, the removal of retained fæcal matter, having been attended to, the second indication, viz., preventing its reaccumulation, must be considered. Gentle tonics, combined with mild aperients, must be resorted to, of which the following is an example: aloes socotrinæ, ext. hyoscyami up to two grains, quinine four grains, ferri sulphate three grains, made into four pills, one, two, or three times daily, or two at night. Aloes is especially useful in the fæcal sluggishness of elderly or sedentary persons, and if there are no hæmorrhoids, and if given in proper doses, aloes may be taken habitually without bad results. The thing is to find out the quantity which will secure one daily dejection. As adjuvants a grain of ipecac may be taken early in the morning, or a quarter of a grain of belladonna extract morning and evening, increased gradually to one grain. Care in diet, and attention to the teeth, so that mastication may be performed satisfactorily, are most necessary.

Constipation may be a result of either acute or chronic *typhlitis*. Acute inflammation of the cæcum may occur either as part of an attack of dysentery, or alone. In the former case it is merged in the symptoms of the general affection of the intestines implicated. But in either case the ulterior results may be the same. Of inflammation of the cæcum three types may be recognised: a catarrhal type which does not end in thickening or ulceration; a more severe type which is apt to leave thickening or ulceration; and a very chronic type which usually ends in the same manner. The catarrhal type more especially attacks children, and

usually subsides after a few days' pain, tenderness, and constipation, under the influence of aperient medicines.

The more severe inflammation most usually commences suddenly, after, probably, a history of dyspeptic derangement, the characteristic symptoms being pain of a colicky nature, and tumours in the right iliac fossa. The right iliac region soon becomes very tender, and to relieve the muscular tension over it, the patient reclines on the right side, with the thighs drawn up. This tumour is usually composed of fæcal matter which is not expelled, resulting in obstinate constipation and vomiting, often of a stercoraceous character. The attack having lasted from two to twelve days, often subsides, the bowels are copiously relieved, the vomiting ceases, and the pain, tenderness, and tumour disappear from the right iliac region. Sometimes, however, the inflammation thus commencing in the cæcum, extends all over the colon, when constipation gives place to diarrhœa and tenesmus, or to characteristic symptoms of dysentery.

The indications of treatment of this form of constipation are to relieve and dislodge accumulations from the cæcum with as little irritation as possible. Saline aperients or castor oil with opiates, large warm enemata, poultices to the part, and a strictly fluid diet are the most generally serviceable. In cases of obstruction, after aperients and opium fail, the former should not be continued, but one grain of morphia, with five of extract of belladonna, may be given twice or three times daily.

It is, however, the chronic form of inflammation of the cæcum which is most frequently met with among Anglo-Indians. The symptoms are at first apparently trifling, and the malady may therefore remain long unrecognised and unattended to, or it may be regarded as the remains of an attack of dysentery, if such has occurred. There is a vague failing of the general health, gradual loss of flesh, and occasional transient colic-like or shooting pain in the cæcum.

Or these transient pains may occur first, without any decided deterioration of the general health. As the malady progresses, which may be during years, there is loss of appetite, flatulence, constipation, and more decided and permanent local uneasiness, or a feeling of fulness and heaviness about the part which on examination may be distinctly felt thickened. There may be occasional darting pain, probably at intervals of days or weeks. These symptoms are relieved for a time by aperient medicines. But at length the habitual constipation alternates with diarrhœa, and eventually the internal coat of the cæcum ulcerates, and there is an aggravation of all symptoms with mucous or slimy discharge streaked with blood as in dysentery, for which the malady may now be mistaken; and sometimes large quantities of pure blood are passed. Broadly speaking, the treatment consists in nourishing and easily digested food, in the frequent application of some counter-irritant as iodine paint, and in the administration of tonics. If the malady is recognised in the earlier stages much benefit will result, and the progress of the disease will be delayed indefinitely by never allowing the bowels to become constipated, and for this purpose a small daily dose of some mineral water is most advisable.

Constipation of Children.—Although infants and children are more liable to diarrhœa in India than in Europe, this does not prevent them suffering very frequently from constipation, which indeed is oftentimes the origin of diarrhœa. The constipation of children depends on different causes, the principal of which are improper food, sluggish action of the liver, and weakness of the muscular coat of the bowels, the latter usually a consequence of a feeble condition of general health. One, two, or all these causes may be in operation, and it is by the discovery of and appreciation of such causes, and by combating them by change of diet and hygienic measures, that the evils arising from the con-

stipation of children may be best prevented. Among these evils may be mentioned fever, convulsions, and spasmodic croup, all of which may and do often arise from hardened fæcal masses in the bowels.

The symptoms vary considerably. In one case there may be simple infrequency and hardness of the motions; in another case the stools are like little dark balls; in a third instance they are white, greenish, or mottled; in a fourth instance they may be accompanied by a little watery or greenish discharge, in a fifth by white or jelly-like mucus; in a sixth they may be streaked with blood, the result of mechanical irritation of the lower bowel.

When the stools are simply infrequent and hard the only indication of ill-health is usually fretfulness or uneasy sleep. But as the stools assume the appearances last noted there is flatulence, fœtid breath, colicky pains, occasional vomiting, often fever, and possibly in female children discharge from the privates. Infants when constipated often bring up their milk in lumpy masses, some of which also passing through the intestines undigested causes increased irritation, flatulence, and colicky pain.

When constipation is long continued, alternating with watery discharges as above noted, the irritation of the hardened fæces is apt to establish a permanent discharge, which may be mistaken for diarrhœa, and treated in vain until the real cause is understood and attacked. This, however, is not true diarrhœa, but as the child becomes more out of health and weakened a true diarrhœa does ensue, which then requires a different treatment.

As a general rule the constipation of infants and children is best treated by some change of food; or, if the infant is being suckled, by some change of food and manner of life of the nursing woman. Generally, she will require more exercise and a larger proportion of vegetables. To afford immediate relief a dose of castor oil may be given to the

woman, which will usually be followed by some temporary diminution of the infant's costiveness; or a little cow's or goat's milk may be given the infant, diluted with half and one-third of pure water respectively, and sweetened to a 'mawkish' sweetness, only with sugar of milk. The temperature should be that of mother's milk, from 96 to 98 Fahrenheit. If the child is being fed by hand, a change from cow's to goat's milk, or *vice versâ*, may be tried. The insertion of a pawn stalk into the fundament may be employed for infants; or a piece of soap may be pared to the thickness of a quill, dipped in salad oil, and introduced into the anus. Rubbing the bowels from above downwards with cocoa-nut oil or salad oil is often efficacious. Injections are to be avoided if possible, although they are excellent remedies in cases of great debility with constipation, and the best material is warm water. When infants suffer from constipation, flatulence, and vomiting of milk in lumpy masses, a mixture containing bicarbonate of magnesia and oil of aniseed, or lime water, will probably be beneficial.

If constipation appears to depend on inactive liver (as denoted by want of colour in the stools), or on want of power in the intestines (usually accompanying a feeble general condition), and it becomes necessary to administer opening medicines to infants or children, the first choice lies between rhubarb, magnesia, castor oil, and senna. If there is costiveness, with flatulence, fœtid breath and acidity, citrate of magnesia may be used. If there is no evident cause but presumed want of intestinal power, or irritation about the cæcum, castor oil. If there is inactive liver, senna may be used. If this is not successful and there are hard clay-coloured or mottled stools, with occasionally watery discharge, a grain of podophyllin may be dissolved in a drachm of brandy and one or two drops of the solution given twice a day on a lump of sugar. Under this treatment there is often immediate improvement. The frequent use of purga-

tives is, however, deprecated. The constipation of children should, if possible, be overcome by change of dieting. A little treacle sometimes answers admirably.

Lastly, when children suffer from alternate constipation and diarrhœa, with fœtid breath and colicky pains, the possibility of worms should be recollected.

CHAPTER IX.

DENGUE.

Synonyms: *Scarlatina rheumatica, dandy fever, three-day fever, red fever, break-bone fever, leg fever, broken-wind fever, eruptive articular fever.* Bengal—*Tootiah.* Rajpootana—*Tup Huddee.* Southern India—*Nudak mariata* (the deity of stiffness of joints). Arabe—*Kidinga papo* (cramps caused by evil spirits).

History.—This disease occurs both epidemically and sporadically in India, Burmah, Arabia, but appears unknown in Europe, excepting in Spain, where a slight epidemic presented in 1871. Something very similar, however, has been described in connection with scarlet fever, both by Drs. Richardson and Golding Bird. In India Calcutta appears to be the most usual *habitat* of the malady, cases occurring there nearly every year. The first epidemic on record was in Calcutta in 1824, when it was called 'red fever.' A severe epidemic occurred in Calcutta and the neighbourhood in 1871–72, when 70 per cent. of the *employés* of Government and of the railways were attacked, and when, as presumed, the proportion of attacks among the general public was even greater. The last epidemic in Calcutta first appeared among the Jewish community, who have most constant and close relations and communication with Arabia. In 1870 it was supposed to have been conveyed by the troop-ship 'Jumna,' from Aden to Bombay. But as sporadic cases occur in Bombay as well as in Calcutta, the theory of importation is not necessary to account for an epidemic spread in either city.

Symptoms.—In the greater number of cases the first symptoms of dengue are headache, restlessness, chilliness, debility, pains in the back, limbs, eye-balls, and joints of a very severe character, and more or less febrile excitement. Sometimes there are not preceding feelings of malaise, the attack of pains in the joints and headache with fever commencing suddenly. Occasionally the first symptom is a sudden pain in a finger. Shortly afterwards, often within twelve hours after the first feelings of uneasiness, sometimes not till three days have elapsed, an eruption of a red or scarlet character appears, lasting about forty-eight hours. The glands of the neck, groin, or axilla, may also become tender; the joints swell; there is generally sore-throat; and occasionally the epidydimis, or testicles, swell. During the eruption the temperature rises to 103°, or even 105°, and the pulse to 120, and at the height of the fever there may be delirium, especially in children, who are also sometimes convulsed. But this rise of temperature only lasts for one or two days, and is not usually indicative of danger, disappearing as the rash disappears, after which, for three or four days, there is almost total remission. Then, with an accession of fever, a second eruption, more resembling that of measles, occurs, first showing on the limbs and resulting in desquamation. Occasionally this eruption resembles nettle-rash. It may be so slight as to escape notice; it may last a few hours or persist for two days.

Both the initial and secondary rash are very variable in character. Sometimes the first is very similar to that of measles, sometimes, as above stated, the second resembles nettle-rash instead of measles. At times both rashes present similar characters; with all varieties of the eruption there is generally intense itching.

The subsidence of the fever, and the disappearance of the eruption, often leave the patient much weakened with rheumatic soreness and stiffness of the joints. But these

after-pains, so common and distressing to grown-up people, seldom cause much trouble to children, who recover much more rapidly and thoroughly than adults.

The period of incubation of dengue is probably from five to six days. In simple and uncomplicated cases the duration is about eight days, but pain and soreness of the joints may remain for weeks or months. Albuminous urine may also be a sequence, but this gradually disappears. There is often obstinate constipation, both throughout the attack and afterwards. It attacks both adults and children, even infants, when the startings occasioned by the pain may be mistaken for convulsions.

Although the typical symptoms of dengue are as stated above, the malady may be much slighter or much more severe. Malaise and trifling pains in the limbs, or slight sore-throat and malaise, often occur during an epidemic, and may be the only symptoms. On the other hand, dengue sometimes assumes a malignant form, when the fever passes into drowsiness and coma, the surface becomes cyanotic, or algide symptoms, resembling the collapse of cholera, supervene. However the malady shows itself, relapses are frequent, persons often suffering from two or three attacks.

As dengue so frequently occurs epidemically, spreading like influenza through cities and over large tracts of country, it is regarded as a highly infectious disorder. If, however, dengue were judged of by the experience I relate below, it might well be asserted to be non-infectious. In the autumn of 1874, when dengue was prevalent in all the villages throughout the country, the camp of the Agent to the Governor-General for the States of Rajpootana—some 1,500 strong—left Mount Aboo *en route* for Agra, a distance of about 300 miles. We marched ten or fifteen miles daily, encamping as usual near the different villages, and no measures to prevent communication were, or indeed could be, enforced. As Superintendent-General of Dispensaries and Vaccinations

in Rajpootana, I was passing daily from the camp to villages, and I constantly saw persons suffering from dengue. But not a single case occurred among the camp people, although there were the usual number of agues and ordinary maladies at the camp hospital. Possibly our life in tents and our moving every day secured that amount of ventilation and fresh air which rendered the poison of dengue innocuous.

The distinctions between dengue and scarlet fever are as follows. Dengue occurs in a much more decidedly epidemic manner than scarlet fever. A high temperature is more rapidly attained by dengue, and it declines more rapidly. The severe muscular and joint pains of dengue are not characteristic of scarlet fever. Dengue does not confer protection against recurrence. The character of the secondary eruption generally differs. Still I believe the maladies are closely allied, if not identical; for in some instances it is quite impossible to distinguish the rash of dengue from that of scarlatina.

Treatment.—As dengue is a specific disease, when once commenced it must run a certain course, and medical treatment has little effect on that course. Still much may be done to alleviate the symptoms as they arise. Constipation should be relieved, and some febrifuge, as the liquor ammoniæ acetatis should be given. Sleeplessness and great pain in the limbs may be combated by Dover's powder or chloral. Tincture of belladonna has been much recommended, and is stated to confer great relief. It may be given in from ten to fifteen drop doses, at intervals of a couple of hours. I have not, however, seen any benefit result from this treatment. Quinine is recommended by most authorities, if the fever, as is sometimes the case, assumes a periodic type. Warm baths, in which a couple of pounds of common washing soda have been dissolved, are useful; otherwise repeated tepid sponging. The cold bath has been advised, with the view of reducing bodily temperature, but would

be likely to check the eruption and so probably do injury. Fayrer recommends camphuretted oil for irritation of the skin. Vinegar and water is also useful. For dengue in children little treatment is required. A senna aperient and cooling draughts of citrate of magnesia will be advisable, and if the child is teething the gums, if hot, tumid, or tender, should be lanced. If there is a tendency to convulsions, bromide of potassium. For the after-pains of dengue, colchicum and alkalies. Recovery is much promoted by change of air.

CHAPTER X.

DIABETES MELLITUS.

YEARS ago diabetes was not regarded as a malady specially affecting the natives of India, Morehead stating only six cases had come to his personal knowledge. However this may have been formerly, there is no doubt that very large numbers of Indians suffer from it now. Chevers believes that among the upper and middle classes of natives in Calcutta, almost every family has lost one or more of its members from diabetes, and in one family nine suffered from the complaint. Dr. Temulji,[1] Bombay, states he has met with many more cases of diabetes recently than in former years; perhaps, he remarks, because the urine is more frequently examined. It is not probable that diabetes is really more frequent among Indians than it used to be. More cases are discovered in consequence of so many more natives coming under skilled medical diagnosis, the result partly of increasing desire among natives to avail themselves of such skill, and partly arising from the large number of Indian practitioners who have been educated in the different colleges.

Diabetes in natives has been attributed to rice-eating. Many, however, live principally on rice and do not suffer. Rice is mainly starch in its composition, containing only from five to seven per cent. of albuminous or flesh-forming constituent. But something more than a rice diet is re-

[1] *Ind. Med. Gaz.*, 1885, p. 223.

quired or the malady would be much more prevalent, and more prevalent among the lower classes in the rice-eating districts than it is. Indolent, obese, well-to-do, perhaps gouty natives, are the class principally affected. Although perhaps not eating meat they take abundance of butter, rice, sweatmeats, and sugar, with little or no physical exercise. They may possibly also take liquor or opium. The process of assimilation of their rich or mainly carbonaceous food and drink is obstructed from want of oxygen which can be obtained by physical exercise only, and which alone can remove the superabundant carbon in the shape of CO_2. Fat accumulates under the skin, and in other parts of the system, producing stoutness as its first effect, and afterwards diabetes, which may be regarded as an effort of nature to get rid of superabundant fat by turning it into sugar in the laboratory of the liver. Mental strain has also been regarded as an exciting cause of diabetes, and Indians engaged in mercantile pursuits, or occupied in office work, are much exposed to worry and anxiety. There is also the undoubted tendency of diabetes to heredity, demonstrated by what has previously been advanced of the numbers of Indians of one family suffering. But the pathology and cause of diabetes is conjectural. It has been ascertained that obstruction to the breathing and other forms of obstruction to the flow of blood, will, as stated by Dr. Pavy, occasion a highly saccharine state of the urine, such as observed in whooping cough, pneumonia, and coma. In healthy persons the blood which passes from the liver to the lungs is saccharine. At its exit from the liver through the hepatic vein, it contains the maximum amount of sugar; after it mixes with the blood from the lower parts it is less saccharine, and as these conjoined currents (in the *vena cava ascendens*) mix in the right auricle with the blood flowing into it through the *vena cava descendens* it is still less saccharine. While the blood passes through the lungs from the right to the left

heart, it loses its saccharine principle altogether, probably from oxidation. It is therefore evident that a function of the lungs consists in the destruction of sugar, the arterial system being thus protected from invasion by saccharine element. When, however, the liver produces sugar in excess, it cannot be destroyed so fast as it reaches the lungs; it invades the arterial blood, and is removed by the only easy mode of egress, that is, by way of the kidneys. Thus diabetes may result as theorised by either an increased production of sugar, or from the diminished destruction or consumption of the quantity nominally produced. This overproduction and diminished consumption depend on an increased ingestion of saccharine material into the stomach and bowels, or on such an alteration of nerve-influence as will modify the relative proportions of the sugar produced and the sugar destroyed.

The following from an official report is typical of diabetes as it occurs in natives: 'A B, a stout, flabby-looking Hindu, a clerk in comfortable circumstances, aged forty, states that twelve months back he noticed frequent and copious micturition of colourless urine, which he ascribed to exposure in a malarious climate. When he came under treatment he was found to be suffering from occasional attacks of intermittent fever of irregular type, with enlargement of the liver, his pulse being weak and tongue red and furred. He was passing 180 ounces of pale urine in the twenty-four hours, containing much sugar, with a specific gravity of 1036 and acid reaction. Appetite voracious, thirst urgent, skin dry, bowels costive, pains in the back and loins; impotent.'

Occasional symptoms are emaciation from the first, but often Indians do not show this emaciation for a long time. The disease may also come on suddenly, but this is rare, for although it may not have been recognised, careful inquiry will usually adduce sufficient evidence to show the malady has been long persistent. Often patients state they were

first suspicious of something wrong by noticing ants and flies being attracted to their urine, others from the quantity of urine voided, others from itching of the genitals, others from impairment of sexual power or wasting of the testicles. There is indeed every reason to believe that Indians often suffer from diabetes for many years without feeling cause for alarm or applying for medical aid. Still in a minority of instances the disease is rapid, leading to early progressive emaciation, exhaustion, and perhaps sudden death. Other occasional symptoms are local perspirations, one limb for instance being moist while the body is dry, or *vice versâ*. A chloroform smell of the breath is often noticed. Not unfrequently there is a foul dry mouth, the teeth being covered with adhesive mucus. The gums may become red, tender and swollen, and the teeth loose, so that the person may at first be thought to be suffering from scurvy, which indeed he is, with the complication of diabetes. Diabetes retinitis, and especially cataract, may also occur. Diabetes insipidus and mellitus may co-exist, and polyuria is more likely when mental exciting causes previously mentioned are evident.

The tendency towards a fatal termination is greater in the young than in the elderly. The most frequent end is by some form of cerebral paralysis, either from hæmorrhage or effusion followed by coma; or by coma produced probably by nervous exhaustion without effusion. The next most frequent termination is by some lung-affection followed by exhaustion. It is sometimes complicated with locomotor ataxy. Carbuncle, nephritis, and cancer may also be regarded as occasional terminations. Diabetes mellitus must be distinguished from that glycosuria of a temporary character which occurs during or after certain diseases, such as cholera, diphtheria, typhoid malarious fevers, or from digestive or nervous derangements, or after some poisons, as morphia, chloral, hydrocyanic acid, mercury, and alcohol. Temporary glycosuria does not lead to the extensive tissue-derange-

ments and general deterioration characteristic of diabetes mellitus.

Diabetic urine is of a pale straw colour, sweet taste, and faint apple-like odour. Thirty or more pints may be passed in the twenty-four hours. The specific gravity varies from 1,030 to 1,060. It often causes heat and burning of the urethra. When kept in a warm place it ferments rapidly, deposits a sediment, and forms quantities of torulæ or fungi, seen under the microscope to consist of round transparent nucleated bodies. The best tests for diabetic urine are Moore's and Trommer's. Moore's test consists in adding half the bulk of pure liquor potassæ to the urine and boiling it; if sugar is present, melassic acid is formed, and the liquid becomes a dark brown. Trommer's test consists in adding a drop or two of sulphate of copper solution to the urine, and then liquor potassæ in excess. On boiling, a yellow brown precipitate of sub-oxide of copper appears. If there is no sugar the mixture becomes green and a black precipitate will fall. The ready-made solution known as Fehling's may be used. But Fehling's solution is affected by heat, and therefore not so well adapted for use in India as in England.

The best *treatment* for Indians is directing them to live as much as possible on anti-diabetic diet, but as many will not take meat, this is the more difficult. Whatever diet is laid down, care should be taken to introduce it gradually, so as not to disgust the patient. Then much may be done by the use of fish, if this also be not against the caste, by gluten or bran cakes, or almond bread, or prepared biscuits, and by green vegetables and green pickles. Cream, skimmed milk, cheese and butter in moderation may also be used, as they are only sugar-formers by virtue of the glycerine they contain. Cocoa made from nibs may also be taken, and tea without milk or sugar, or with the smallest addition of the latter. Most fruits are forbidden, but oranges, Indian grapes, and pummaloes may be taken in moderation. On the other hand, there is the

avoidance of all articles containing sugar and starch. Claret and water is the best drink, and if any spirit is allowed it should be sound whisky, which contains less sugar than any other liquor. Thirst may be relieved by washing the mouth out with iced water, which is better than copious draughts. Lemon water, or lemon sliced in tea, will also relieve the thirst and cleanse the mouth. As medicines, tonics will be advisable. Salicylic acid in fifteen-grain doses sometimes does good, and small doses of opium are generally beneficial. Constipation is best relieved by small quantities of Carlsbad salts or Janos water. Warm clothing is a *sina quâ non*, as the diabetic are very liable to chills and chest-affections. Warm salt baths and change of climate to the sea-side are also advised. The weight of the patient should be taken frequently, for the increase or decrease indicates the efficacy of the treatment or the reverse. Exercise should be taken to as great an extent as possible short of fatigue; and massage, or shampooing the limbs, seems beneficial, probably by increasing metabolism.

CHAPTER XI.

DIARRHŒA.

Every form and description of diarrhœa is met with in India, but the ordinary varieties may be classed as follows:—1. Diarrhœa premonitory of cholera. 2. Diarrhœa premonitory of dysentery. 3. Diarrhœa symptomatic of organic disease, principally of the liver and spleen. 4. Irritative diarrhœa. 5. Anæmic diarrhœa. 6. Diarrhœa from defective hygiene and sanitation. 7. Nervous or mental diarrhœa. 8. Diarrhœa from atmospheric vicissitudes. The first three forms are sufficiently noticed under the heads of the respective diseases of which they form a part.

4. Irritative Diarrhœa arises from the direct irritation of the intestines by, *first*, excess of food, or by food of improper quality, and must be regarded as an effort of the intestines to rid themselves of offending matter. The result may be an attack of headache, perhaps accompanied by retching, or an attack of colic, characterised by griping pains sometimes accompanied with nausea, faintness, and violent vomiting of bilious material, succeeded by copious stools. A frequent cause of this form of irritative diarrhœa is the use of stale or deteriorated tinned provisions, which when decomposing dissolve the tin, with which they become impregnated, or the use of copper cooking utensils from which the coating of tin has worn off, when the food becomes impregnated with copper.

Secondly, irritative diarrhœa may result from impure

drinking-water, most usually from that containing mineral matters and commonly called 'brackish.' When persons pass from districts in which the water is good, into perhaps the more numerous localities in which the water is brackish, they frequently suffer from diarrhœa, especially at first, and particularly if no precautions are taken with regard to boiling and filtering the water. Vegetable impurities in water have been also credited with inducing diarrhœa, but no one has been able to state what these impurities are, unless under the general term *malaria*, which may mean anything or nothing.

Thirdly, irritative diarrhœa may arise from the excessive secretion and elimination of bile, and this again results in a so-called bilious attack or bilious colic, characterised by the symptoms mentioned above.

Fourthly, retained fæces may induce a condition of alternate constipation and diarrhœa (*vide* p. 144), and especially so in children (p. 148).

Fifthly, there is the condition known as lienteric diarrhœa, in which from indigestion food passes into the intestines in a more or less unchanged state, and acts as a direct irritant.

Sixthly, there is diarrhœa caused by the irritation of worms.

5. Anæmic Diarrhœa arises from that blood-deterioration considered under anæmia, and may also result from the scorbutic condition, from leprosy, and other blood-disorders; also from want or famine. D. Cunningham has shown that a process of chronic starvation induces desquamative changes in the intestines of a fatty, lardaceous and atrophic nature, which render them unfit for the functions of nutrition. The essential process appears to be deposit of lardaceous material and softening.

6. Diarrhœa from Defective Hygiene and Sanitation.—That diarrhœa may arise from damp, cold, unhealthy dwellings, emanations from decaying material, from foul

drains and sewers, &c., is undoubted. But unless the cause or causes as above are prominent and powerful, it is generally impossible to decide how much any particular diarrhœa is due to defective local sanitation, and how much to anæmia or other causes, many of which are generally in action at the same time.

7. Nervous or Mental Diarrhœa.—This form of diarrhœa is probably more frequently seen in India than elsewhere, partly owing to the general tendency to bowel complaint which exists in the East, and partly to the anæmic or scorbutic taint from which so many suffer, lowering the tone of the nervous system. It is known that irritation of the sympathetic nerve increases the activity of the glands of the intestinal canal, and also the peristaltic action of the small intestines. In any climate both an acute and a chronic or intermittent diarrhœa may be excited or maintained by worry or anxiety. The intestinal nerve-centre sometimes becomes so sensitive that every meal excites an action of the bowels, time not being allowed for digestion or absorption. Such diarrhœas have been termed *lienteric*, but this only signifies the type of the discharge and not the cause, which is nervous. The conditions favouring this nervous diarrhœa are exaggerated in India, where so many have onerous work and responsible positions, their state of health being below par. This diarrhœa is one of the predisposing causes of cholera.

8. Diarrhœa from Atmospheric Vicissitudes.—This is the most common form of diarrhœa in India, and should be denominated climatic. I include under this heading those diarrhœas spoken of as 'malarial' as hill diarrhœa, and to a certain extent the fluxes known as *vicarious* and *lienteric*. I believe the most common cause to be atmospheric changes, producing chill, acting on a cutaneous surface, predisposed to chill from debility caused by heat, and in many cases also acting on a constitution predisposed by an anæmic or latent

scorbutic condition of blood, probably accompanied, as Surgeon-Major O'Connel[1] has shown, by an excess of water in the blood. I consider the term malarial diarrhœa as unnecessary, and not in accordance with our absence of knowledge whether there really is a specific malarial poison. I do not see, when diarrhœa arises from chill causing suppressed perspiration, or from chill causing suppressed renal secretion, or from 'renal inadequacy' from any cause, or from pulmonary embarrassment, it should be termed 'vicarious,' the real causes being chill, or the other conditions referred to, and the diarrhœa therefore not being vicarious in the proper sense of the word.

The slightest degree of tropical diarrhœa from atmospheric causes is that which occurs especially during the rainy season, in the early morning on rising, or on first passing out into the open air. Some individuals, especially if at all anæmic, may be said to be scarcely ever free from a slight morning diarrhœa, two or three loose evacuations, accompanied with perhaps slight griping, occurring every morning. In such cases it may, within certain bounds, be regarded as salutary, just as when it follows imprudence in diet, for which form of diarrhœa it is very usually mistaken. Another slight form of diarrhœa, which has been usually ascribed to malaria, occurs after a person has been actively engaged during the day—perhaps snipe-shooting in the heat of the sun. He returns home, feels a little feverish, has diarrhœa during the night, and in the morning feels well again. This depends on chill and fatigue, or unaccustomed exercise, and is not to be attributable to a specific poison, but rather to exposure when tired to the evening fall of temperature, or to a dense shade after the skin has been acted upon by a vertical sun. Again, diarrhœa often results from sudden changes of temperature, as occur, for instance, at the commencement of the Indian monsoon; and although

[1] *Ind. Med. Gaz.*, 1884.

such changes may not be so great as those happening in some other climates, it must be recollected that the changes which do occur have cutaneous and general systems to act upon debilitated by great and continued heat. Also that persons in India from the mode of life, such as open doors and thin clothing, are more exposed to chill than persons residing in a temperate climate. Exposure to damp night air, damp clothing or bedding, sleeping in draughts, are all fertile sources of chill and consequent diarrhœa. If to any of these causes of atmospheric diarrhœa are added the causes previously named—anæmia, defective sanitation, &c.—the disease is aggravated and the diarrhœa is more likely to progress. It may indeed be stated that the most prevalent and fatal diarrhœa of India especially among the natives, is the effect of climatic, of local causes, and of careless exposure.

Diarrhœa originating from either of the causes named may be *ephemeral*, lasting from hours to a day or two; *acute*, continuing from ten days to a fortnight; or *chronic*, persisting for an indefinite period. The diarrhœa may also be of two kinds, viz., diarrhœa *biliosa* and diarrhœa *alba* (white diarrhœa); the former chiefly prevailing in the southerly districts of India, near the sea-coasts, and in the hot season, although common everywhere and at every period of the year; the latter seen principally on the more elevated plateaux, and at hill stations, and in the colder seasons. The diarrhœa biliosa has been called *thermic* diarrhœa, and is marked by dark-coloured, often offensive, frothy stools, accompanied by griping pains, flatulence, nausea, furred tongue, and burning pain in the rectum, and the onset is often sudden and may be accompanied by nausea and vomiting.

When this diarrhœa is very acute and accompanied by prostration or cramps, and if cholera prevails, the probability of its passing into the latter disease is great, for there is

certainly a connection between the two maladies, as is shown by the great prevalence of diarrhœa during cholera seasons, when it has been noticed that the habitually constipated become the reverse. The presence of bile or undigested food in the stools, and the absence of suppression of urine, are the principal diagnostic features. From dysentery *diarrhœa biliosa* is known by the absence of blood or slime in the evacuations, by the less amount of tormina and tenesmus, and by the absence of tenderness about the sigmoid flexure and colon.

Diarrhœa alba, from its prevalence on the Himalayan hill stations, has been termed 'hill diarrhœa,' and from its being so frequently noticed at Simla in former days acquired the vulgar name of 'Simla trots.' At this time, however, Simla was almost the only hill station where a number of Europeans were congregated, and therefore the malady was most noticed there, leading to an erroneous idea that it was a special malady of the locality. Years back Grant stated that few persons on the Himalayas escape one or more attacks in the course of the season, and that the malady was not confined to persons of broken-down health, or who had suffered from fever, but that residents in sound health and in the prime of life were affected. *Diarrhœa alba*, however, is not confined to the hills, but is a common malady in all the more northerly plains of Hindustan.

The *symptoms of diarrhœa alba* are usually painless, but sometimes painful, purging occurring at first, principally in the morning. The stools may at first be bilious-looking, but are often from the commencement, and always afterwards, light, often almost white in colour. They are also copious and frothy. As the disease advances light stools are also passed in the evening, but the patient probably continuing to feel tolerably well takes little notice of the commencement of the malady. The calls to stool, although generally unattended with pains, are urgent, but the fæces

are passed without straining or faintness, and are succeeded by a feeling of comfort. The most annoying symptoms are fulness and distension of the bowels by flatus, and eructations having the flavour of rotten egg. At first the stools themselves are not offensive, although afterwards becoming very much so. Often no abnormal sensation is felt in the region of the liver, but sometimes from the very commencement there is an uneasy sensation. When this is inquired into it is found to be rather a feeling of void than fulness, as if the ribs were about approximating. As the disease advances the appetite, at first good, becomes the reverse, the pulse grows more feeble, the tongue is furred in the centre, and faintness may follow the stools. There is sometimes at a later period some degree of sallowness, and a bronzing, similar to that in Addison's disease, has been noticed. If the malady is not checked the person falls into a state of confirmed cachexia. The stools become more numerous, progressive emaciation takes place, the mind becomes weak and fretful, and fever may occur. Then, probably, the stools become dysenteric and the patient dies exhausted.

The stools, tested by nitric acid, sulphuric acid and sugar, and acetate of lead, often do not give the least evidence of bile, but fatty material may be extracted. The disease is marked throughout by a progressive deterioration of the secretory apparatus of the intestines, with a tendency to shedding of the epithelium with granular and fatty infiltration. The urine is frequently natural throughout, sometimes showing traces of bile.

Diarrhœa alba on hill stations has been attributed to numerous causes, the principal of which is, of course, the general noxious agent, malaria. Grant, Farquhar, Murray, and many others have supported this theory; Surgeon-General Murray adding to malaria the effects of neglected conservancy, for which Simla was long notorious; others have attributed it to bad drinking-water (Parkes), especially

at Dhurmsala, where the water contained fine scales of mica. But a consideration of facts tends to show that the disease must be ascribed to the sudden lowering of temperature experienced by those visiting a hill station, or to sudden changes experienced on a mountain range. This view is supported by the fact frequently noticed of change of colour of the stools immediately on ascending a mountain, often within a few hours. Also by the absence of fever or other disease usually attributed to malaria, which has caused those supporting the malaria theory to presume that malaria too diluted to cause fever is blown up from the ravines, where 'dhobees,' washing all day in the sun and sleeping out all night, often get colic; but which is explainable from their mode of life, without malaria. *Diarrhœa alba* also prevails mostly in the autumnal season, when the vicissitudes of temperature and damp are greatest. Again, new arrivals are more subject than older residents, who are well housed and know how to take care of themselves. Ewart and Fayrer say it may occur in persons who have not shown any symptoms of suffering from malarious influences, but that it most frequently happens in the progress of convalescence from malarious fevers, and they may have added, at the time the system is more than ordinarily liable to be influenced by vicissitudes of temperature. Lastly, there are some hill stations, as the Neilgherries and Mahableshwar, where the disease is very seldom seen. On Mount Aboo also it is never of the inveterate, almost epidemic, character which it has presented on the Himalayas, and the reason is apparent. The latter are inter-tropical hill stations, and the temperature is both higher and more equable than on the Himalayan stations, which are extra-tropical, and which, with a hot sun, are exposed to icy winds from the snow-clad regions to the north. Hence the visitor from the plains below to the inter-tropical stations undergoes a less sudden and a less frequent variation of temperature than he

does when visiting or sojourning in the northern regions of the Himalayas.

The true pathology of *diarrhœa alba* would appear to be torpor of the secreting cells of the liver, probably the result of chill. It is not at variance with physiology to presume that nervous influence may be lessened by cold or vicissitudes of temperature acting on the sympathetic, and especially on one of its ramifications, the hepatic plexus. A somewhat similar condition results in other parts from the effects of cold land winds, when, after exposure, an irritable bladder becomes powerless to act, or when from the same cause a limb becomes benumbed and powerless.

Under the above views it is easy to interpret the various attendant phenomena. Bile being the natural purgative, why should its absence cause diarrhœa? Jaundice not taking place, what becomes of the colouring matter? Bile, or its principles, being evidently retained in the system, why do not head-symptoms arise similar to those sometimes supervening on jaundice? The fact, however, of diarrhœa occurring both from excess and deficit of bile is not so paradoxical as it at first appears. Anything irritating the intestinal tube will cause diarrhœa. There are, as the effects of the absence of bile, imperfect digestion of albuminous substances, imperfect emulsion of fatty matters, the non-neutralisation of irritating acids, the loss of the antiseptic property of the bile, and nothing to prevent the accumulation of viscid mucus on the coats of the bowels. All these abnormal occurrences must lead to irritation and diarrhœa. Jaundice not taking place, and no bile passing off by the excretions, points to the conclusion that no bile is formed, and secondly strengthens the conclusion that bile is altogether formed in the liver. Head symptoms do not occur, because no bile is present to excite them. The blood becomes depraved, but the poisonous matter of bile must be absent when none is manufactured.

There are, therefore, reasonable grounds for regarding *diarrhœa alba* to be the result of impairment of hepatic nervous power, leading to the absence of bile and consequent diarrhœa. The resulting cachexia and wasting is attributable, not to the absorption or retention of bile which has never been formed, but to the want of those constituents of the bile in the system which are normally absorbed.

Those who, like Inman, maintain that the brown colour of the stools is not due to bile, but to some secretion of the colon, have regarded white diarrhœa as a result of a disordered condition of the latter organ. This position is fortified by several ingenious arguments. In health it is stated the brown colour of the fæces begins after they have entered the colon, and the nearer they are to the duodenum the lighter is the colour. This is correct; but on the other hand, yellow fluid is often found in the jejunum and ileum, and the loss of colour of the bile is due to mixture with the pancreatic juice, which is an almost colourless liquid. That the material becomes dark again after entering the colon is due partly to absorption of lighter coloured matter, and partly to exposure to the greater amount of gas, and to the peculiar gases generated in the large intestines. It is also stated that in obstruction of the biliary ducts the intestinal secretions remain the same colour throughout the bowels. But complete obstruction rarely does occur.

It has been stated that scrofulous persons are most liable to *diarrhœa alba*, which may be questioned. The scorbutic taint will, however, predispose to *diarrhœa alba*, as to any other form of bowel complaint.

Post-mortem appearances.—Bowels pale and blanched, coats of intestines atrophied, mesenteric glands and mucous follicles enlarged, mucous membrane soft with serous exudations beneath, inflamed spots on the peritoneum, liver pale, hepatic abscess rarely concomitant. When death has occurred at an early period from some other disease, the intestines

have been found contracted, with the mucous membrane thickened and corrugated; but when death has occurred later the coats of the bowels are attenuated with lardaceous degeneration of the glandular structure. Similar degeneration has been found in the liver and spleen. Ewart and Fayrer say the liver and kidneys are often found in a state of chronic inflammation.

Treatment.—The treatment of the diarrhœas described under the terms irritative, anæmic, from defective hygiene, nervous, and *biliosa* may be surmised from the names. If diarrhœa is due to irritating contents of the intestines, aperients will be necessary. A dose of castor oil and opium will often suffice. Anæmic diarrhœa is only to be benefited by measures to combat the cause of the anæmia. A similar remark applies to diarrhœa from defective hygiene. In nervous diarrhœa the irritability may sometimes be allayed by the bromides, and nervine tonics, such as nux vomica or oxide of zinc, are often efficacious. Opium or chloral may be used cautiously. But usually the habits and manners of life of the person suffering from nervous diarrhœa must be changed.

In the treatment of the diarrhœa described as *biliosa*, if the stools are very dark and offensive, and the tongue foul, and if there is no cholera about, blue pill with ipecac and hyoscyamus may be given. Afterwards Dover's powder combined with quinine is recommended. But astringents and opium should not be used too early in this form of diarrhœa. Great attention should be paid to diet, discarding all solid food, and the recumbent posture, warmth to the bowels, and avoidance of motion should be insisted upon.

As regards the treatment of *diarrhœa alba* the diet must be specially attended to, and as the intestines are incapable of assimilation, food rich in materials for the formation of blood and tissue, which may be mostly disposed of by the stomach and upper bowels, should be mainly relied on. Of

these animal broths or jellies, eggs and Liebig's raw meat soup are the best. Milk is also advisable and often suits well, but if undigested caseine appears in the stools, it should be given up. A similar remark applies to milk and suet. A good deal depends on the quality of the milk, which must not be in the least soured. Sometimes milk agrees best after boiling. Farinaceous gruels or pudding may be cautiously used, but often they do not suit. Stimulants are usually required from the first, of which a little port wine is best. As a drink, water with white of egg is advisable. Dover's powder five grains, combined with quinine five grains, and ipecacuanha one grain, should be administered night and morning. If the ipecac produces sickness, the quantity to be reduced or omitted. A mixture composed of chloroform one drachm, aromatic spirits of ammonia one drachm, brandy one ounce, a tea-spoonful in water every three or four hours, is often grateful and beneficial. At the same time measures should be taken to induce the liver to act. Mustard poultices or leaves should be applied daily, or as often as can be borne, both over the right hypochondrium and over the bowels. Small doses of podophyllin should also be used. A grain should be dissolved in a drachm of brandy, and six or eight drops of the solution given twice a day on a lump of sugar. Euonymin may also be tried. If these means do not produce the desired effect, an occasional dose of blue pill, the milder preparation, grey powder, not being reliable in India, as owing to the chemical changes it often becomes deteriorated, and sometimes even poisonous. Astringents of any kind are not recommended. In very anæmic cases nitro-muriatic acid with taraxacum may be employed. The flatulence with rotten-egg flavour may be much mitigated by a drop of creasote or carbonic acid on sugar. Change of climate is, however, the best measure.

Chronic Diarrhœa.—All kinds of diarrhœa in India may become chronic. Digestion, absorption, and assimila-

tion are interfered with, and wasting from inanition results. The diarrhœa increases in consequence of the increasing debility, and thus a vicious circle is established, cause aggravating effect, and effect aggravating cause, and probably the spleen becomes enlarged, or dysenteric symptoms may supervene. Careful dieting will in chronic diarrhœa be more beneficial than any kinds of medicine. Beef-tea, beef-extract, chicken, veal, or mutton tea, broths and soups, chicken jelly, fine mince, flour and milk, Liebig's raw meat soup, panada, and farinaceous puddings form a sufficiently wide choice. Much attention must also be given to the avoidance of chill. A routine prescription of astringents is to be avoided. If there is marked mental depression or nervous anxiety the bromides may be cautiously used. If food passes unchanged, as in *lienteric* diarrhœa, arsenic has been found useful. Generally tonics are advisable, the most useful of which is quinine with sulphuric acid.

But diarrhœa of any kind occurring in India if long continued requires change of climate, which should be taken before the patient becomes greatly debilitated. The remarks on this head made under chronic dysentery are applicable. A severe chronic diarrhœa requires at least two years in England, even if the person is apparently well soon after arrival, as the disease is very likely to return on re-entering the tropics. It is in these cases of chronic diarrhœa that the 'milk cure' is frequently beneficial, and it is more efficacious if carried out in Switzerland.

Diarrhœa of Infants and Children.—The diarrhœa of infants is most commonly caused in the order named by errors of diet leading to undigested food and acidity, or accumulation of hard fæcal matter in the bowels, by atmospheric changes, damp and cold, by teething, by worms, by tubercular disease of the bowels; or it may come on during whooping cough. The diarrhœa of children is often accompanied by acid vomiting and sour breath, is always

attended by more or less flatulency, and frequently by griping, evidenced by the straining cry of the child and by its legs being spasmodically raised towards the bowels when pain occurs. An infant's bowels should be relieved three or four times daily, and the motions should be of the colour of mustard, and free from fœtor or acid smell. When the stools are more frequent, but of a natural colour, and there is no fœtor, the diarrhœa is probably caused by an accidental error of diet, or by atmospheric vicissitudes, and it may be regarded as of comparatively little importance. When the stools are yellow but becoming green after exposure it denotes a large secretion of bile, and there is still little cause for anxiety. When they are green or greenish-yellow, with sour smell and containing specks or flakes like bread-crumbs, or larger masses of white curdy material which may be undigested milk or mucus from the bowels, there is much internal irritation present. When they are white an inactive liver is denoted. In children, especially when hand-fed, there may be clay-like stools containing masses of undigested caseine, which after evacuation become greenish from contact with concentrated acid urine, converting whatever little colouring matter of the bile may be present into green *biliverdin*. When diarrhœa of a watery character alternates with constipation, the latter may probably be the primary cause of the mischief. When diarrhœa comes on suddenly, the stools consisting altogether of greenish coloured water accompanied by much exhaustion, the condition approaches that of cholera.

As a rule diarrhœa in children should not be too suddenly checked, particularly if the child is teething, when it is frequently a salutary effort of nature to relieve the irritation of the system thus excited. If the purging is moderate, the colour of the stools natural, and the motions at all consistent or formed, it will most usually subside without any medicine. But the diarrhœa of infants and children in India

cannot be permitted to run on without treatment so long as would be warrantable in a temperate climate. Even the mildest form should not go unchecked longer than twenty-four hours. For it must be recollected that the rapid exhaustion of the vital powers of a child caused by diarrhœa is very favourable to the supervention of such maladies as hydrocephalus, convulsions, or dysentery. The first thing to do is to look to the food, with the view of correcting any error of diet. In the case of infants, diarrhœa is often caused by improper food, or by over-feeding, or by some deleterious property of the milk. Infants should not be nursed oftener than every two hours, and as the age advances the periods should be lengthened. If fresh milk is taken into the stomach while some of the last milk remains, the result is generally either purging or vomiting. When the milk of the nurse is at fault, it will probably be due to improper diet of the woman, and this may require not only alteration of the food, but the action of a purgative dose. The milk of nurses is often rendered too rich, or their system is disordered by the desire of their employers to feed them well, and who give them more and richer food than they have previously been accustomed to.

When medicines are necessary the safest treatment is to give at the outset, but not afterwards, a little castor oil, which will relieve the bowels of any irritating material. If the purging continues, chlorodyne may be given in doses corresponding with the age of the child. If the child is feverish at one time and cool at another, quinine with Dover's powder. If the breath or stools smell, lime water. If there is vomiting of curdy material, milk previously boiled should be tried. If the child passes large offensive curdy stools, the milk should be stopped, and Liebig's raw meat soup, or weak chicken broth, should be given for two or three days. Both in the diarrhœa and dysentery of children, such a change of food is often attended with much benefit when-

ever the motions show that milk is not being digested. When the milk is resumed it should be well diluted, and lime water should be added to each meal. If there are white stools alternating with constipation, the purging will not cease until the liver acts, for which a grain of podophyllin may be dissolved in a drachm of brandy, and one or two drops of the solution given twice a day on a little white sugar.

Sudden diarrhœa, with greenish watery stools and great depression, should be treated as cholera. The chloroform, brandy, and ammonia mixture mentioned at p. 173, given in doses according to the age of the child, is one of the best remedies for this sudden diarrhœa of children.

When the above remedies and care in diet do not prove efficacious, a mixture composed of sulphuric acid twenty minims, tinct. catechu forty minims, syrup of ginger two drachms, aqua ten drachms, is a good medicine in cases of simple diarrhœa, to which if there is much griping laudanum may be cautiously added, one small drop for each year of a child's age being the ordinary dose. While thus giving medicines for the diarrhœa, the gums should be examined and lanced if necessary.

The preceding refers to *acute* diarrhœa, but the diarrhœa of children if neglected often becomes *chronic*. There are five or six pale or variegated offensive motions daily, occasionally varied by watery discharge, while the child becomes thin and wastes. In such a condition the temperature should be taken twice daily. If it is that of health there is probably little the matter, but if it is persistently higher there will be cause for anxiety, especially if the form of diarrhœa has succeeded some other illness, when the commencement of tubercular deposit in the intestinal glands may be feared. When such cases occur great attention must be paid to protection from damp and cold, to the ventilation of the sleeping apartment, and to careful regulation of the diet. Potatoes, sweet biscuits, farinaceous foods

generally, also sugars, and jams, should be interdicted. Bread and milk, a little fresh meat, green boiled vegetables may be allowed. Some one of the malt foods now prepared should be given, and peptonised milk may also be tried. The great point, however, is to get the liver to act, and for this purpose podophyllin dissolved in brandy may be used (*vide* p. 177). But persistent diarrhœa in a child generally eventually requires removal from India, although change of locality may be first tried.

The diarrhœa of infants and children may in any climate pass by almost insensible gradations into the condition known as *muco-enteritis*. (*Vide* Compendium.)

CHAPTER XII.

DRACUNCULUS.

Synonym :—Guinea-worm.

THE literature of the subject is enormous, even extending from the time of Moses, as there seems reason to believe that the so-called fiery serpents which attacked the Israelites during their sojourn in the wilderness were dracunculi.

It is essentially a tropical malady, but it is not confined to India, having been noticed in Egypt, Arabia, Central Asia, Abyssinia, Guinea, and Brazil. According to Rudolphi's nomenclature it is called hair-worm (*Filaria medinensis*), but this name does not convey a good idea of the parasite, as it is much thicker than hair, appearing like a thin piece of catgut, one-eighth or one-sixth of an inch broad, slightly elastic, of a milk-white colour, and semi-transparent. In some stages of its growth the extremities for an inch or two are as thin as hair, but become suddenly thicker. The anterior extremity, or head, is recognised by a punctum in the centre, and round it are minute rugæ, external to which are two papillæ, one on either side. But it is difficult to obtain the head for examination, as it is usually rubbed off or becomes disintegrated. On each side (*i.e.* dorsal and ventral) there is a longitudinal, probably muscular line, and the worm is also marked by numerous transverse *striæ*. The body of the worm contains an alimentary canal, which commences at the punctum and terminates in the concavity at the tail end. The genital organs consist of a large sac or tube,

terminating abruptly at either extremity in a much smaller tube. The whole extent of this uterine sac or capsule is crowded with innumerable young, the whole seeming to be a uterus or *proligerous capsule*, in which Bastian asserts young are produced 'without love, or courtship, or matrimony, as the result of a non-sexual process. However this may be, it is certain that all which have been found are female worms, and that males are not known to exist in the human body or elsewhere. Cuvier stated the worm acquires the length of ten feet or more, but the average length of a number of specimens preserved by the late Dr. Grierson (Bombay Army) was 23⅝ inches. The average length of a number examined by Assist.-Surg. Minas of Bombay was twenty-three inches. It may, however, attain a much greater length, for I have seen one three feet long. On breaking the adult worm the young are poured out, appearing under the microscope very like extremely minified dracunculi.[1] H. Carter found that minute worms (*Urobales palustris*), having great resemblance to the young of the guinea-worm, existed in abundance in some of the ponds and wells near Bombay, and supposed they might penetrate the integument *viâ* the sudoriferous channels, and so grow into guinea-worm. The position was supported by the fact of the boys of the Byculla schools suffering from guinea-worm, while others who did not, as the boys did, use water from wells containing these worms did not suffer. In support of the theory Sir Barrow Ellis,[2] then Revenue Commissioner, Bombay, remarked that guinea-worm was most prevalent among those who used water from wells to which a flight of steps led down, and instances were recorded where the same people having obtained water from a well without steps became free from the disease. As no male guinea-worm has been found in the human system, it was also theorised that the slime and débris collecting on the steps of wells is the nidus in which the guinea-worm

[1] *Bombay Med. and Phy. Soc. Trans.*, 1853. [2] *Ibid.*, 1862.

breeds, when the male perishes, while the female penetrates or is taken into the human body. This view appeared to receive further support when it was found (by Messrs. Duncan and Forbes) that the young of the dracunculus do not die when placed in water, but live about six days, and when placed in moist clay twenty days. But it was pointed out at the time that there is a very considerable difference in size between the young of the guinea-worm and that of the worms found in tanks. Other observers, as Dr. Greenhow (Bengal), failed in finding these tank-worms, although searching in localities where guinea-worm was prevalent. I also drew attention to the fact that guinea-worm was common in the semi-desert districts of Marwar and Bickaneer, where water is hundreds of feet from the surface, where there are no steps to wells, and consequently where there is no slimy nidus for the development of the worm. Experiments, by inoculation and swallowing these tank-worms, have failed to propagate the disease. Still more recently Cobbold, on the strength of the representations of the Russian Fedschenko, has advanced that the escaped embryos perforate the skin of minute aquatic crustaceans (*Cyclops*), where they undergo further development. At the expiration of a month they attain their highest larval state of growth within the cyclops, and along with the intermediate hosts they are conveyed as male and females to the human stomach. After impregnation in the stomach or intestines, the females migrate while the males pass out with the fæces. But these minute aquatic crustaceans have not been found in various localities where guinea-worm is well known. The fact is, we are ignorant of the life cycle of the guinea-worm, and also as to the mode in which it obtains entrance into the system. It is not impossible that the guinea-worm enters the human body both by the mouth and by the external integuments, and that in both instances water is the medium by which it is conveyed into contact with the body. Its connection with

water is sufficiently demonstrated by the fact already mentioned (p. 5), of the almost total disappearance of guinea-worm from the city of Bombay following the closure of the wells, and the supply of water from the distant hills. That the ova may enter the system by mouth appears probable from the occurrence of the worm in Europeans, who do not, like natives, dabble in or about tanks and wells. I recollect one European gentleman contracting guinea-worm, who always bathed in warm water, and Minas mentions the case of a man who never used water for ablution during a lengthened stay in Bombay, but who suffered from the parasite. That the process of digestion will not suffice to destroy existence is sufficiently clear from the fact of the gad-fly attaining maturity attached to the splenic extremity of the horse's stomach, from instances of leeches, caterpillars, and larvæ of flies having been discharged alive, from trichinæ, &c.

To the conclusion that the ova may make way through the surface, I am led by the fact that there are some 3,500 ducts in every square inch of skin, that the usual position of the worm is the foot or ankle, which are most exposed, from observing that sailors employed on water parties, or who have gone bare-footed about sodden ground around wells, or shooting water fowl, have suffered afterwards from dracunculus.

The late Dr. Scott of Bombay, while asserting the external origin of guinea-worm, stated that the men employed as water-carriers (*bheestees*) are often infested with the plague in their shoulders, on which the water-skin rests; but I cannot adduce this as an argument, not having observed *bheestees* to be more subject to guinea-worm in the shoulder than other persons.

Number of worms observed.—Although the guinea-worm is usually solitary, two or many may present at the same time. Clot Bey records an instance where twenty-eight appeared in the same person. Carter records a case where twenty-seven issued from the right leg and sixteen

from the left. Dr. A. Farre mentions that as many as fifty have been met with in the same person. Minas gives an account of a patient who died, in whom the whole skin was a network of guinea-worms.

Seat or locality.—As already mentioned, the lower extremities are most usually affected, to the extent of 98 per cent. But the worm may be present in any part of the body. Thus, it has been observed in the socket of the eye (Scott), between the heart and pericardium (Morehead), in the cavity of the abdomen, in the liver, in the back and loins. The fact of the dracunculus appearing in such positions seems against the idea that the worms previously mentioned are the cause of the disease, as something smaller than these worms, which average $\frac{1}{32}$ of an inch in length, would be more likely to make way from the point of entrance through the minute blood or lacteal vessels. It must not be forgotten that guinea-worms have exhibited migratory powers. Many cases of the kind have been recorded by Paton, Morehead, Steward, &c. Ewart[1] has seen the worm change its position from the upper part of the lateral aspect of the thorax to the groin in the course of twenty-four hours, but has never seen it travel from below upwards. Of the large number of guinea-worm cases I have seen, not one changed its position, and I therefore regard such change as very rare.

The period of incubation of the guinea-worm is very variably stated, nothing certain being known on the subject. It has been asserted that the guinea-worm never makes its appearance before the second season of residence in the places where it is endemic. This I do not believe, having known the worm developed within five weeks of the arrival of a European in Bombay. Instances have occurred of persons leaving India and only noticing the worm after a four months' voyage round the Cape. Abdoollah Khan, the native doctor of the Ajmere Dispensary, who had great

[1] *Ind. An. Med. Sci.*, vol. vi.

experience of guinea-worm, and who personally suffered, believes the period of incubation to be between one and four months. The period of latency is doubtless commensurate with that of growth—quicker in some cases, slower in others. It probably lies between one and twelve months.

The season of prevalence in India seems, from the reports of various observers, to differ in distant parts of the country. Speaking generally, it is most prevalent during the rains, and next immediately before or after the rains in the months of May and September. Attempts have been made to couple the prevalence of guinea-worm with the dryness of the tanks and the scarcity of water. But, in the Bombay and Matoongha districts, the worm becomes more common in May and June, but it chiefly prevails in the rainy months of June, July, August, and September. Attempts have also been made to connect its prevalence with the period of irrigation of, and work in, the fields. But, as the period of the incubation of the worm is unknown, all such attempts must be failures.

Geological features of soil or locality do not appear to exert much influence. It has been stated that all the districts where dracunculus prevails are composed of secondary trap-rock—*i.e.*, of igneous formation—and that where the parasite is rare the soil is a conglomerate iron-shot clay (Carter). I have, however, mentioned that guinea-worm is common in Western India, where the geological structure is sand on sandstone. It also prevails at Pertabghur, where the soil is apparently conglomerate iron clay of a red colour, and it presents on black cotton soil.

Symptoms.—It is most prevalent between the ages of twenty and forty-five, but it occurs at all periods and in both sexes. Attention is, in most instances, first drawn to its presence by a corded substance felt beneath the skin, like a tense rolling vein. The entozoon generally lies just beneath the true skin embedded in the cellular tissue, but

it often penetrates between the muscles, and, in some instances, is coiled round them or their tendons. As the head of the worm approaches near the surface a small blister forms, and indeed this bullæ, which sometimes attains the size of half an egg, oftentimes attracts the first notice. Some authors state that if this blister be rubbed or broken, it frequently happens that the most insufferable itching occurs. But this I have heard made the subject of complaint before the vesicle bursts, as also eruptions resembling nettle-rash. The formation of the vesicle is in some instances preceded by a pricking pain. Others state there has been no pain. If the vesicle is opened early it contains a clear limpid fluid; if later, there is a thick white mucous. This vesicle, which was formerly regarded as an ordinary blister, is now asserted to be the extremity of the proligerous capsule dilated with fluid, and which escapes through the head of the worm. The latter may be found in the serum of the vesicle, if looked for early, before the serum becomes turbid and the head of the worm has died and become disintegrated. In most instances it is extremely difficult, and often quite impossible, to secure the thin and fragile head. If, however, the attempt is successful, the whole worm may sometimes be secured. If the attempt is not successful the worm is broken, with discharge of large numbers of young worms. If the end of the worm is thus ruptured, and the fluid of the vesicle is removed, a red ulcerated appearance presents, with a slightly depressed conical-shaped hole, in the centre of which are several white spots, which eventually coalesce, and through which the parasite issues. Notwithstanding the breakage of the head of the worm, or even of the body, the death of the parasite does not always succeed. It even seems to be still possessed of a recuperative power, for a second smaller delicate transparent *cul-de-sac* or apparent blister may again present, which appears to be the healed inflated extremity of the

proligerous capsule. This is more likely to occur if the parts are exposed to the influence of running water. When the guinea-worm breaks, inflammation is often caused, which may terminate in tedious suppuration, sinuses, irritative fever, sloughing, gangrene, and may even cause tetanus. Such terminations occur more frequently in those of anæmic habit. I have frequently known the guinea-worm broken without any bad results, as above. It is not, as has been supposed, the discharge of young only which creates inflammation; it is when the parasite dies and acts as a foreign body that inflammation and abscess occur.

Lastly, instances are known where a guinea-worm has apparently dried up or become partially absorbed, giving no further trouble during life, although traces have been found after death.

Treatment.—The plan of making an incision over the worm, passing a probe or ligature underneath it, and gradually extracting, has been often successfully practised, but is open to several objections. It seems probable the parasite is only sufficiently loose for extraction when its head appears at the surface. If the worm is situated altogether in a fleshy part the operation may succeed, but if the reverse, or if coiled round tendons, inflammation may be excited, or the worm may be broken by endeavours to extract. It appears rather the province of the surgeon to assist the natural mode of exit.

When the vesicle before mentioned forms, and appears on the point of bursting, it may be opened, and the thin extremity of the worm sought for. If this can be seen it should not be seized with a pair of forceps, as has been advised, for it is more likely to break than not under contact with the steel, but it should be caught by a roughened feather stem, or other small light rough substance, round which it may be gently manipulated. It occasionally happens that not only the thin extremity, but a little of the worm itself, or in very exceptional cases the whole of the parasite, may be extracted at once. Usually, unless a little

of the body of the worm can be wound round the feather-stem without the slightest force, it will be better to remain satisfied with having secured the thin end of the worm. The feather stem should then be attached to the part by a strip of adhesive plaster, and the whole covered with lint soaked in a strong solution of alum. This hardens the texture of the worm, while still keeping it soft. In twelve hours or so, a cautious attempt may be again made to extract a little more. But much care must be taken lest the worm break in the process of extraction, or lest the part of the worm round the feather becoming dry break without the application of any force. Extraction should only be attempted once in twenty-four hours after the first time, when perhaps an inch, perhaps a foot, may be gained. The force applied should not be great, and the pulling should be delicately and yieldingly managed, the worm as obtained being wound round the feather. It requires a sort of 'knack,' only obtainable by practice, and which some native doctors possess to perfection. A stream of water over the part will often assist extraction. Stories are related of persons with guinea-worm going to running water, holding their foot therein for a time, and so getting rid of the enemy. Such rapid escape of the parasite I have never seen. In cases coming under my observation, days, or oftener weeks, have elapsed before the worm was extracted. If, however, a stream of water is available, it should be used for two or three hours daily, and slightly warm water is best. The exit of the worm may also be facilitated by slight friction with oil, and light pressure over the line of the worm. Occasionally a spontaneous protrusion of a large piece of worm occurs, which is very favourable. If when the vesicle bursts or is opened the end of the worm cannot be seen, it may sometimes be raised by the application of a small cupping-glass. Indian doctors use suction by the mouth through a trumpet-shaped tube for the same purpose.

If the worm breaks, the best treatment is a stream of

water running from above down the line of the worm, and washing the orifice so as to keep the sore clear of young dracunculi. This may be continued for an hour or so several times daily. In a day or two, in all probability, so much of the body of the worm will protrude that it may be seized with a pair of forceps. This is a very delicate operation, and unless enough of the worm comes out on the slightest force, so that it may be fixed to a quill, it will be better to defer the securing till next day. Much irritation is often produced, and much harm is often done, by abortive attempts to seize and drag out the worm. In such instances inflammation is created, and the worm probably dies, thus acting more decidedly as a foreign body, while the minute young passing from the ovisac into the cellular tissue, doubtless add to the fever, tumour, and suppurations which so often ensue. When the breaking of the worm is followed by inflammation poulticing is best, any matter forming being liberated by the lancet.

The natives use many remedies for guinea-worm, among which poultices of the leaves of the *melia azadirichta*, or 'nimb,' are supposed to possess great efficacy. The madar-leaf (*Asclepias gigantea*), tobacco, datura, ghee, and the 'singhy,' or native cupping, are also used. But the treatment as above is the most successful. When the worm is not secured no application will favour seizure; when the worm is secured the point is to prevent it breaking by being kept too moist or too dry, and for this an alum lotion, with oiled silk covering, are the best applications.

Assafœtida and other drugs have been supposed to possess a prophylactic power when used internally or externally, and oiling the limbs is sometimes resorted to as a preventive. But cleanliness and the use of soap with water are the best means of preventing the entrance of the parasite through the integument; and the habitual use of boiled and filtered water of avoiding taking the ovum internally.

CHAPTER XIII.

DYSENTERY.

Is a disease known from the earliest ages in India, Charaka having described six varieties. In modern times many more have been mentioned under such terms as acute, adynamic, asthenic, bloody flux, catarrhal, chronic, contagious, endemic, epidemic, fibrinous, hæmorrhagic, hepatic, insidious, malarious, putrid, scorbutic, sloughing, typhoid, &c. It has also been termed metastasis of rheumatism to the bowels. But these terms merely express phases or complications of the disorder, and no definite line of demarcation really differentiates one type from another, for the disease may commence under one aspect and terminate under another. Forms of dysentery have been described as diphtheritic and croupous, the same distinctions being drawn as between ordinary diphtheria and croup, viz., that the former implicates the structure of the mucous membrane, the latter being simply a deposit on its surface. When these conditions occur in dysentery the diphtheritic formation may pass away in patches, while the croupous membrane may pass as tubular casts. But I do not regard either of these conditions as dysentery, but look upon them as something superadded —as the diphtheritic or croupous growth prevailing in or on a part already prepared for its reception by a diseased state.

The disease may be defined as inflammatory action, involving some part of the large, and occasionally of the small

intestines; often, but not always the local expression of a constitutional disorder.

Although it is in India and in hot climates generally that dysentery is most prevalent, it occurs in colder climates (as in Norway in 1859, when it spread like cholera), especially among armies, garrisons, and fleets, as the records of modern campaigns, from the Peninsular to the American and Franco-German wars, sufficiently show; in all of which dysentery was a scourge, although owing to better general sanitary arrangements, food, &c., a decreasing scourge to the troops engaged in operations. It is, however, in cantonment or civil life that the mortality from dysentery has been most reduced. Thus in India in the European army the mortality fell from 81 per 1,000 in 1859, to 54 per 1,000 in 1860, and has since averaged some 47 per 1,000 of those attacked. This has been accomplished partly by better sanitation and personal hygiene, but chiefly, as most authorities assert, by improved methods of treating the disease.

The classes most liable to dysentery in India are Europeans generally, and European soldiers in particular; the men to rather a greater extent than the women, excepting pregnant females, who are most liable, the disease generally causing miscarriage. The European soldier compared with the sepoy is peculiarly liable—eleven of the former being admitted for one of the latter. But natives, both soldiers and others, suffer considerably, to the extent among the military of 82 per 1,000. Up to thirty years of age there is an increase of admissions among Europeans, then gradually diminishing, but the rate of mortality increasing with age. Up to the tenth year of residence in India dysentery is most prevalent, after that period the proportion of admissions gradually decreases. The teetotaler and the intemperate are affected in nearly equal proportions, and the temperate are least liable. The chances of recovery are in favour of the teetotaler.

The disease is most prevalent in the south of India, especially on the coast districts, becoming gradually less prevalent, although an ordinary malady, as the north of India is approached. Fayrer mentions there are some places in India and Africa where the disease is unknown, but I am unacquainted with such localities.

Season and weather have a manifest influence, the disease occurring to a greater degree in the cold and rainy seasons.

The causes of the disease are variously stated, and opinions are divided as to whether there may be a specific dysenteric poison or not. Since the days of Annesly many authors, as Grant,[1] Hare,[2] Cameron,[3] Armand,[4] Gordon,[5] and Fayrer,[6] have attributed dysentery to malaria. Gordon states dysentery with or without disease of the liver is the result of malarious poisoning, and Fayrer remarks, 'Most cases of dysentery in Bengal at least are more or less associated with malaria.' Ewart[7] states, 'There is an intimate connection between the existence of malaria and the prevalence of dysentery,' and he remarks when fevers have been extinguished by improved drainage, and the conversion of marsh into cultivated land, dysentery becomes equally unknown. If by malaria is understood general climatic conditions, then I agree with the authors named; but if by malaria a specific poison is implied, then I agree with a late writer[8] that 'There is nothing to establish the assumption beyond the fact that dysentery and periodical fevers are both diseases of a tropical climate.' The fact is, that the term dysentery covers a variety of differently caused conditions, agreeing only in the fact of blood in the stools. Thus a dysenteric condition has been ascribed to chill arising from

[1] Grant, *Ind. An. Med. Sci.*, vol. i. [2] Hare, *ibid.*, vol. 2.
[3] Cameron, *Lancet*, 1861. [4] Armand, *Pathologie Internie.*
[5] Gordon, *Report on Typhoid Fever*, 1878. [6] Fayrer, *Tropical Diseases.*
[7] Ewart, Quain's *Dictionary of Medicine.*
[8] Roy, *Ind. Med. Gaz.*, Sept. 1883.

sudden changes of temperature; to imprudent change of clothing, especially that worn over the bowels, or from lying on damp ground; to irregularities in diet; to improper food, especially containing decomposing albuminates, or rancidity of the fatty substances; to famine and want; to drinking marsh water, or water containing animal or vegetable impurities; to fæcal accumulation in the large intestines; to irritant poisons; to residence in ill-ventilated, imperfectly drained, and badly located habitations; to the effluvia from sewage, foul drains, or privies, and from bilge-water on board ship; to intestinal worms; to salt provisions, especially the salt meat of military and naval life, which is often more highly salted and kept longer, and therefore less nutritious, than the salt meat of civil life; to the scorbutic condition; and to blood-poisoning from almost any cause. It would be easy to adduce numerous instances where the disease apparently originated from any of the causes named above. Dysentery often occurs in the East when there is no evident cause beyond general climatic conditions, or, as most regard them, malaria. Everyone practising in the tropics will have met with dysentery following, and evidently attributed to, cold or chill. The usual termination of famine and want is dysentery; while the exhalations from sewage have, even in a temperate climate, caused an epidemic of the disease.[1] It would therefore appear that dysentery may be a local disorder, as when caused by chills or irritant or poisonous matters in the intestines, in the same sense as pleurisy, or tonsillitis, or arsenic-poisoning, are local disorders; or it may be the local manifestation of a constitutional disorder, as when it arises from an anæmic or scorbutic condition, or from blood-poisoning from foul air, or from the violent congestion of the abdominal viscera caused by the cold stage of paroxysmal fevers. Thus some authorities distinguish catarrh of the bowels, although accompanied by bloody stools, as a non-con-

[1] Coulston, *Med. Times*, June, 1865.

tagious local disorder, or a simple inflammation of the colon accompanied by fever; regarding dysentery as a contagious disorder arising from a diseased state of the blood affecting the colon, accompanied by fever and by a characteristic odour of the evacuations. Intestinal catarrh may, however, under favourable circumstances develope into so-called contagious dysentery. When the disease has occurred in crowded barracks, transports, ships, jails, besieged garrisons, beaten and retreating armies, &c., it has been considered due to contagion. Cullen, Pringle, Lind, and all older authors considered its contagious character to be beyond question. Parkes states it is well known that dysentery may spread through a troop from the fact of the same latrines being used; and it has been theorised that the dysenteric poison enters *per rectum*. The effluvia arising from the bodies and fæces of numerous patients in a contracted space will undoubtedly predispose to that blood-deterioration favourable to a local manifestation such as dysentery, but that any specific poison originates there is no evidence to prove. In most of the conditions mentioned above, when dysentery has been referred to contagion, other deteriorating influences have been in activity, such as depression, want of proper diet or clothing, and especially exposure to chills, which are sufficient to account for disease without enlisting contagion as a cause. It is curious that authors who deny the possibility of a *de novo* origin of a cholera poison do not hesitate to teach the origination of the contagium of dysentery. The facts are, however, as stated by Fayrer: 'We do not know how it originates. . . . Whether due to direct infection intensified by concentration, or to a generally depressing effect causing a condition of blood-poisoning I know not. . . . We are not yet in a position to define with certainty the exciting cause of dysentery.' All that we know with certainty is that the immediate cause of most cases of dysentery may be traced to

a chill, and that anything depressing or debilitating the system renders a person more liable to chill.

Symptoms.—Dysentery may be preceded by symptoms of indigestion and general malaise. Or it may occur in connection with an attack of ague. But it commonly occurs without any recognised preceding symptoms. The first signs are feelings of griping about the navel, often accompanied by nausea. Very frequently this is felt after incautious exposure to night air, particularly during sleep, and more especially if the wind has been suffered to play on the bowels even if well covered; or after the incautious use of a tatty or punkah; or after the punkah coolie having gone to sleep, suddenly wakening, pulls the punkah violently over his sleeping perspiring victim. Next there are irregular loose discharges from the bowels, which may continue two or three days—the premonitory diarrhœa of dysentery. The irregular griping pains gradually become more cutting and shooting, with great heat about the rectum, and frequent straining and purging. The matters voided consist first of liquid fæces streaked or mixed with blood, and afterwards of clear tenacious viscid mucus streaked with blood, and often without trace of fæcal matter, especially when the colon or rectum are chiefly affected. The desire to go to stool is generally most urgent during the night, in some instances almost incessant, in others twenty or more calls in the twenty-four hours. In weakly subjects prolapse of the rectum may occur from the straining. When the rectum is affected there may be difficult micturition, or even retention. The patient feels weak, has no inclination for food, the tongue is furred, there is clamminess of the mouth, and the patient often feels chilly. The amount of attendant fever is very variable, in some cases hardly exciting attention, in others evidenced by a flushed face, dry skin, and hard, quick pulse. In favourable cases the disease may subside in from three to eight days; the pains subside, and the stools lose their dysenteric

character. But in other less favourable cases, in addition to the griping pains there is more or less general pain in the abdomen, not the acute tenderness of peritonitis, but rather soreness and fulness, yet distinctly aggravated by slight pressure, especially in the iliac regions. As this tenderness increases, and the disease becomes more severe, generally about the seventh or eighth day, shreds or patches of mucous membrane pass away, which in some degree resemble washing of raw meat, and are best seen when the discharges are mixed with water. In some instances tubular pieces may pass, but it is rare in modern times to see those terrible cases of sloughing dysentery which, according to older authors, were once so common. When tubular sloughs present, they are generally diphtheritic, or more probably croupous exudations. But it is believed that occasionally tubular portions of mucous membrane do pass; also that sloughs of sub-mucous tissue may pass, much in the same way as occurs in bad forms of erysipelas.

Urgent and continued tormina and tenesmus, strangury or dysuria, pain in the cord and testicles, absence of pain and tenderness in the bowels or iliac regions, and mucous or bloody stools unmixed with fæces, point out the rectum as chiefly implicated. The passage of shreds or sloughs, coupled with a cadaverous fœtor, pinched and anxious countenance, harsh dry skin, hiccough, and involuntary motions, pronounce the case hopeless, and death may take place from cardiac asthenia. In other cases the mischief may spread to the peritoneum with the usual results of peritonitis, viz., great local tenderness, and effusion of serum and lymph. Or ulceration may give rise to copious hæmorrhage, and when blood is observed in large quantities unmixed with mucus ulceration may be diagnosed. It should be recollected that ulceration may take place during the first few days, although most frequently occurring after the disease has prevailed a longer period. Gangrene may also set in, indicated by cold,

clammy sweat, sunken eyes, husky voice, thready pulse, cold limbs, absence of pain, muscular tremors, and horribly fœtid stools with black sloughs resembling old sooty cobwebs. 'In gangrenous dysentery the pains suddenly cease, the patient thinks he is going to recover, smiles a sickly smile, and dies' (Chevers).

Peculiar symptoms.—Constipation with scybalæ in the bowels are occasionally the premonitory symptoms, instead of diarrhœa. Dysentery has been observed to commence sometimes with a discharge of blood, which is stated to be most common in Bengal, and not to depend on an early ulceration. An insidious form has been described by Matthew, in which there is offensive diarrhœa for some weeks, followed by sudden bleeding from the rectum with absence of abdominal pain or febrile excitement, from which it is argued that ulceration must have progressed without the appearance of dysenteric discharge. An aphthous condition of the mouth is often seen when diphtheritic or croupous conditions prevail internally. Dysentery sometimes assumes a periodic form, and is associated with distinct attacks of fever.

Complications.—1. Scorbutic dysentery. The scorbutic taint, although it may be latent (*vide* Scurvy, Chap. XXVII.), plays a far more important part in the origin and aggravation of dysentery than is in the present day usually imagined, and if any existing scorbutic condition is not recognised and made the subject of treatment, all medication will be futile. It may be admitted that scorbutic dysentery is not so common among soldiers as in former years, owing to the better dietary now supplied, but it is still sufficiently common, especially among sepoys, who have a habit of depriving themselves of sufficient diet to save money; also among the civil population, many of whom, especially in seasons of scarcity, are unable to procure proper food; also among the inhabitants of the semi-desert districts, who cannot obtain

a sufficiency of fresh vegetable diet. Scorbutic dysentery was formerly very common on board ship, but now, owing to shorter voyages under steam and better food, it is seldom seen. But in the year 1855, I had an opportunity of seeing a number of cases of scorbutic dysentery on an American ship in the Persian Gulf, among the crew of which scurvy had prevailed during the voyage round the Cape. The type answered to the putrid dysentery of Lind and Clarke. It may be recognised by the livid complexion of the surface generally, by the petechiæ, by the spongy, bleeding gums, by the discharges containing grumous blood, by œdema of the feet or more general anasarca, and by the termination of the disease, which is sometimes by hydrothorax, or more frequently by ascites, as in one form of scurvy (*vide* Scurvy, Chap. XXVII). The causes of so-called scorbutic dysentery are precisely those of scurvy, of which it is in reality a form.

2. With anæmia it is often associated, assuming in such cases a low, asthenic form.

3. Also with intermittent and remittent fevers, when the dysentery may be more violent and intense; or may assume the low, asthenic form which characterises it when occurring during severe anæmia. It is to these phases of the disease that the term 'malarious dysentery' has been applied, and it is these phases, in connection with fever, which assume the so-termed 'contagious' form.

4. There is reason to believe that the form of dysentery described as *bilious* is sometimes mistaken for typhoid. It sets in with diarrhœa and yellow stools, and there is fever of the remittent type, accompanied by great debility and severe headache. There is often nausea, and sometimes vomiting. As the case progresses the evacuations become dysenteric. Eventually there is great restlessness, irregular, quick and soft pulse, dry and brown tongue, and other symptoms of the typhoid condition. This form of dysentery prevails chiefly among weakly persons in so-called malarious localities,

or in those affected by latent scurvy as a consequence of scarcity.

5. Dysentery is frequently a termination of elephantiasis, and of leprosy, and must therefore be due to the same causes.

6. Hepatic Dysentery.—In dysentery complicated with liver disease the cæcum and rectum are found chiefly affected. In 196 cases the small intestines were affected in twenty-six, the cæcum in eighty-two, while perforation occurred in twenty-three, and thirty-nine were complicated with liver abscess; out of which thirty-nine cases in twenty-two the disease chiefly affected the cæcum. That a large number of fatal cases of dysentery are complicated with liver-abscess is an established fact. Some years back, when Assistant-Surgeon to the European General Hospital, Bombay,[1] I collected the experience of twelve observers who varied from a percentage of 50·9 to 8·5 of abscess accompanying or following dysentery. But the percentage on 1,532 cases of all observers amounted only to 18. Even at this lower ratio it would appear that liver-abscess is much more commonly associated with dysentery in the East than in Europe, or even in semi-tropical climates, for the records of the Mediterranean comparatively seldom show any connection; and of numbers of soldiers who came under treatment for dysentery in the Malta Hospital only two were the subjects of hepatic abscess (Marston). Dr. Baly's testimony was to the same effect as regards the dysentery occurring some years back in the Millbank Penitentiary. There must therefore be something different in the state of the liver in the East Indies, rendering it more liable to be affected. Dr. Budd first originated the opinion that liver-abscess complicating dysentery was due to absorption of vitiated matter from the diseased intestines; and others have regarded it as caused, not by direct absorption but by systematic poisoning, or to pyæmia; and this view has

[1] *Bombay Med. and Phy. Soc. Trans.*, 1862.

been strengthened by the occasional result of pyæmia after operations about the rectum when liver-abscess has been found. I am, however, disposed to believe that the coincidence is not to be viewed as result and cause, as in practice it is found those most prone to suffer from the liver during dysenteric attacks are individuals who have suffered from some liver-malady previously, or who have been long in the country. I think both dysentery and liver-abscess are produced by general common climatic causes, and that while such causes excite dysentery in one, and liver-abscess in another, they often also excite a combination. However this may be, the frequent occurrence of abscess with dysentery is indisputable, and the practical effect of our knowledge should be the insisting on a prompt, accurate, and daily exploration of the hepatic region. It is not always easy to distinguish hepatic complications from an inflamed and ulcerated transverse colon; but if, during the progress of dysentery, rigors, sweats, persistent rise of temperature, or bulging appear, or if during convalescence from dysentery the emaciation and languor are greater than can be accounted for by the existing dysenteric symptoms; if evening hectic, with tongue red at the tip and edges present, there is little cause for doubting that hepatic abscess has become established.

The seat of localisation of dysentery is chiefly in the large intestines, but in some instances, and especially when there is a scorbutic taint, it may extend into the small intestines for some inches above the ileo-cæcal valve, and the whole tract may be in a catarrhal or even diphtheritic condition, an adventitious membrane having been found even in the stomach (McLeod). The lesions are the results of inflammation and exudation affecting the mucous and submucous tissue, and particularly the glandular structure of the gut. According to Parkes and Cornil, the disease commences in the glandular structure, but according to other observers it commences either in the glandular structure

or in other tissues. Whether it commences in one or the other, there are four clearly defined processes of ulceration : *First*, hyperæmia; *second*, deposit of exudation; *third*, expulsion of exudation by ulceration, and *fourth*, cicatrisation. Chuckerbutty described no less than thirteen kinds of ulcer or slough, which however simply indicate different phases or stages of the disease. Other authors describe *minute*, *irregular*, *serpentine* or *rodent*, *transverse*, *circular*, *oval*, *tubercular* ulcers. Some of the ulcers were carefully observed at the European General Hospital, Bombay, and at least three modes of formation were seen. First, there are seen some enlarged solitary glands causing slight prominences of the mucous membrane, and accompanied by a few minute red vessels. Others, a little larger, showed an umbilicated depression, and in the next stage the mucous covering at the depressed point becomes a rugged ulcerated opening, which increases in size until the enlarged gland is fully exposed and then apparently falls out, leaving an ulcerated pit, which, as the disease progresses, increases in size. A *second* mode seems to be by destructive or suppurative inflammation in the *subjacent* tissue, the mucous membrane over the spot sloughing and leaving a foul surface exposed, the disease of the areolar tissue undermining the mucous membrane at the margin of the ulcers, giving them a peculiar appearance of elevated rings. A *third* mode is that in which the process appears to begin in the mucous membrane. The surface of the colon shows circular ulcers of various sizes and mostly with red margins, also red spots of different degrees of intensity. These points increase by extension of ulceration, and the sub-mucous areolar tissue is laid bare. In some spots the destruction is so entirely confined to the mucous membrane that the denuded tissue forms, when the ulcer is yet small, a prominence in its middle, which on a cursory glance may be mistaken for a large and denuded gland.

Thus the autopsy may reveal vascularity, vivid inflammatory redness, ulceration sometimes perforating the abdominal cavity, or sloughing, either throughout the intestinal tube, or confined to a certain portion, or scattered here and there with spaces presenting a healthy appearance; or at a later date commencing cicatrisation may be evident. Micro-organisms are to be seen in abundance in those cases in which the disease has advanced to ulceration and sloughing, their presence being indicative of parts undergoing devitalisation, as in gangrene, erysipelas, &c., and therefore not specific.

When cicatrisation occurs, it is matter of doubt if the intestinal mucous membrane is ever entirely reproduced, and I am inclined to think this depends on the depth of the ulcerative process. Often after sloughing great puckering and narrowing of the canal has been observed, and sometimes the gut by measurement has been found to be shortened.

Treatment.—In former days bleeding and mercury were the principal means used, the former, as Robert Jackson directed, 'until all that was expected of it had been obtained;' the latter, as Pringle, James Johnson, and Annesly recommended, until the patient was salivated. Gradually, however, the anæmic tendency of all disease in India began to be understood, and partly on this account, and partly in consequence of the theory of the change of type of disease originating with Allison and Christison, and lessening the amount of bleeding at home, the use of the lancet ceased to be the inevitable custom in India, and was relegated to patients with good constitution and not long in the country; while large doses of calomel, such as ten grains at bedtime, took the place of salivation. Both Morehead and Peet, for instance, recommend ten grains of calomel at bedtime, and give certain symptoms as guides to the repetition of the dose: and this, although Morehead states the practice of salivation has been most properly abandoned, and Peet would not give

mercury to asthenic patients. As with the calomel, so with the bleeding, it was only as the elder medical officers retired, and younger men took their places, that the system was discontinued; for from April 1, 1858, to March 31, 1860, in the European General Hospital, Bombay, ten cases of dysentery out of thirty-three treated in one set of wards were bled to the average of 12½ ounces; and nineteen out of forty-nine treated in another set of wards were leeched to the average amount of twenty-five ounces. Although bleedings and calomel were thus still used by the older practitioners, the following, written by myself years ago, gives a view of the usual practice some quarter of a century back: 'In the mildest form of the affection, when griping pains are complained of at intervals, followed or accompanied by the discharge of slightly bloody stools, fomentations, rest in the horizontal posture, demulcent drinks, and a pill every three hours composed of ipecacuanha one grain, blue pill two grains, and opium half a grain, will frequently effect a cure in a few days. In the more acute forms of dysentery, when the calls to stool are very frequent, the pain cutting, the abdomen tender, and the constitution good, the application of a large number of leeches to the abdomen is called for, while calomel should be substituted for the mercurial before mentioned; the recumbent posture, hot fomentations, and demulcent drinks being insisted upon.'

Probably a somewhat similar system would have been continued had not Mr. Docker[1] (2 batt. 7th Fusiliers) in 1858 reported great success in the Mauritius by the revival of the use of large doses of ipecacuanha, called of old the *radix anti-dysenterica*. I say a revival of practice, because Bontius in 1629 recommended for the treatment of dysentery—bleeding being premised or omitted as judged necessary—ipecacuanha, by way of a vomit at the beginning. In 1662 the drug was employed in France by a physician

[1] *Lancet*, July, 1858.

named Lequais on his return from Brazil. We have it also recorded that Piso treated a son of Louis XV. successfully by large doses of ipecac. According to Cornish, it was again used in India in 1795 by Dr. Graham, who was told the use of it by an African friend. Clarke in 1770 used large doses with antimony. Afterwards it was lauded by Balmain,[1] was used by Abercrombie 1807,[2] and by Playfair 1813.[3] It was also used by Twining and Pringle in smaller doses. But it fell into disuse until Mr. Docker's report, when the practice was taken up by Cornish in Madras, by Ewart in Calcutta, and by others. In 1861 Cornish[4] published a lengthy paper on the use of large doses of ipecac in dysentery, and embodied the opinion of thirty-three medical officers who practised the method in obedience to a circular from the head of the Medical Department. Of these, twenty-two spoke decidedly as to its specific efficacy, eight spoke less enthusiastically, and three rather condemned it. Peet also, in his book published about the same time, did not speak well of it. Ipecac also proved useless in the epidemic of dysentery occurring in 1864 in the Cumberland Asylum.[5] But notwithstanding failures, the ipecac treatment of dysentery became the established practice, and, owing to the writings of Ewart especially, and others, has maintained its reputation. So early indeed as 1868 it was stated: 'Thanks to ipecacuanha, the mortality from dysentery is everywhere reduced. Prior to the commencement of the last decade it was upwards of 11 per cent., and now below 5.'[6] Shortly after the revival of the practice I wrote, 'The general advice is that the large dose should be preceded by opium, which it is said prevents its nauseating and so debilitating the patient. In the cases in

[1] *Mem. Med. Soc.*, London, vol. v. p. 210.
[2] *Ind. Med. Gaz.*, Sept., 1860. [3] *Ed. Med. Surg. Journal*, vol. ix. p. 18.
[4] *Madras Quart. Jour. Med. Sci.*, No. 111.
[5] Coulston, *Med. Times*, June 10, 1865.
[6] Francis, *Ind. Med. Gaz.*, June, 1868.

which I have used this remedy it has certainly checked the discharges and pain in a wonderful manner; but, owing to the nausea and distress it occasions, it is difficult to prevail on some patients to continue it. If they can bear it, the treatment may be depended upon in acute cases; but in chronic it affords very temporary relief, scarcely in correspondence with the distress it occasions.' And this may be regarded as applicable now. Fayrer states, 'Acute dysentery in previously healthy persons if dealt with early, before the bowel has passed beyond the stage of catarrhal congestion, is most amenable to treatment,' by ipecac, but 'when the disease has advanced to ulceration, and when the characteristic stool has been fully established, ipecac is no longer useful.' Maclean also notices an intolerance to ipecac in cases complicated with liver-abscess. As showing the benefit of ipecac given at once in acute cases, McLeod may be quoted, who writes, 'A man was taken ill on Saturday, had his ipecac on Saturday night, and was able to attend office on Monday.'

The approved method of treating acute dysentery is to give forty drops of chlorodyne in a tablespoonful of water, and fifteen minutes afterwards thirty grains of powdered ipecacuanha in a wineglassful of water, and then to apply a mustard poultice over the epigastrium, not the bowels, for twenty minutes. The patient should lie down with the head low, and remain perfectly quiet and refrain from drinking, but if thirsty he may suck ice. This will probably cause nausea and depression, but it does not do so in all cases, and the after-result is generally free action of the skin, subsidence of griping, and reappearance of natural stools. If vomiting does not occur, or if it does not occur in a very violent manner, and if the patient is not very depressed, the same medicine should be given in about eight hours afterwards, and repeated if necessary at such intervals during three days; care being taken to allow of sufficient time between the doses to admit of the patient taking and

digesting some fluid nourishment. If the ipecac is not well borne, it will be advisable to alternate the doses with soap and water injections containing forty grains of the powder. I prefer chlorodyne to an opiate to precede the ipecacuanha, as this preparation, although not pharmacopœial, is equally efficacious in preparing the stomach as opium, and exerts no after-effects.

Some difference of opinion exists as to the propriety of using ipecacuanha when the patient is decidedly anæmic, but Fayrer states it is better tolerated than might be supposed even by the anæmic, and he recommends a cordial before and after the dose, while Maclean states that in the subacute stage debility does not forbid. But I do not think the toleration of ipecac depends nearly so much on the strength or weakness of the patient as on the temperament and idiosyncrasy; for there is scarcely any drug towards which idiosyncrasy is more displayed, and this is probably the cause of ipecac being borne so badly in numerous, although perhaps exceptional, cases.

To pregnant females, again, it must be given with caution. It is an observation as old as the days of Bontius, that 'When pregnant women are seized with tenesmus, they generally miscarry.' But a miscarriage may also be caused by vomiting, and this is most likely to happen if the patient is anæmic. On the other hand, the ipecacuanha may cut short the disease, and so prevent a miscarriage which will certainly occur if the dysentery goes on unchecked. Therefore for dysentery occurring in pregnant women, the ipecac treatment is the best that can be adopted, but it must be employed cautiously. In dysentery complicated with pregnancy, Ewart states opiate enemata, to relieve irritation in the rectum, are more essential than under other circumstances.

The manner in which ipecac acts is not to be regarded as specific. It has been attributed to the power of 'revulsion,' to 'a sedative action on the sympathetic nerve, whereby an

antiperistaltic action of the intestines is effected;' but it seems to me the good result should be attributed chiefly to the action on the skin. Be this, however, as it may, it is regarded by Ewart as producing 'all the benefits that have been ascribed to blood-letting without robbing the system of one drop of blood, all the advantages of mercurial and other purgatives without their irritating action, all the good results of antimony and other sudorifics without their uncertainty, all the benefit attributed to opium without masking the disease.'

Now, fully admitting good results from the ipecac treatment which Maclean and others state has reduced the mortality from dysentery fully one half, I am still not disposed to credit ipecac to that extent. For the fact is, there has taken place a reduction of mortality from nearly every disease, which must be attributed not only to improved medical treatment, but also to the causes mentioned at p. 4.[1] A diminution in the actual number of cases of dysentery has taken place both in Bombay and Calcutta since the provision of better water, while the type is less severe, and the mortality, irrespective of treatment, must therefore be less.

As before mentioned, the vomiting and depression produced by ipecac are sometimes so great that the treatment cannot be continued. In such cases, or when, as sometimes happens, ipecac fails to prove beneficial, it will be advisable to give one grain of ipecac, five of Dover's powder, and three or four of quinine every four hours; the quinine being especially required if the patient has been in a malarious district, or if there is accompanying fever. Fayrer observes that most cases of dysentery in Bengal are associated with malaria, and therefore quinine is a desirable adjuvant to other drugs, and may be given to cinchonism to prevent fever. Quinine indeed was given by Hare to the extent of eight scruples

[1] For a consideration of the whole subject *vide* the Author's papers on 'New and Old Medicine,' *Ind. Med. Gaz.*, 1878.

daily, but these large doses are not absorbed, and are therefore not required. It would scarcely be imagined that a disease essentially inflammation of the interior of the bowels could ever have been treated by purgatives. Yet it has been argued that as dysenteric stools do not contain fæces, the disease is a constipation, and treatment by purgatives has been sanctioned by such experienced officers as Pringle, Twining, Bampfield, and others. It is, however, unsuccessful in practice, if not erroneous in theory, and now deservedly abolished. I do not, however, wish to be understood as objecting to purgatives *in toto*. Stating them as a rule to be inadmissible, I am yet fully aware that it is sometimes necessary to relieve a tumid abdomen at the commencement of a dysenteric attack. Indeed, I make a point of inquiring if the bowels have been confined previously, and if evidence of retained excretions exists, I commence the treatment with the most emollient of all purgatives, castor oil, perhaps combined with tincture of opium, and would use the ipecacuanha afterwards.

In the advanced stage of dysentery, when there are ulcers or sloughing known by the characters of the motions (*vide* p. 195), ipecacuanha is not recommended. The object is to give rest to the bowels and to support the strength, and to avoid everything liable to irritate the intestines. Fayrer recommends twenty drops of oil of turpentine with ten drops of tincture of opium three times daily, with local medication, but states recurrence of acute symptoms indicates the need of ipecacuanha. But in such cases, after the primary time for ipecacuanha has passed away, I am more disposed to trust to chloral given in small doses with the view to its calmative effect only, to good nursing, and care to maintain the recumbent posture (a recommendation as old as the days of Celsus), with nourishing diet, which is best obtained by limiting the patient to good milk or milk boiled with a little flour. If febrile symptoms prevail,

quinine may be used. If there is any reason to suspect a scurvy taint, chlorate of potash with excess of lime-juice, or plain lime-juice and water as a drink, with the pulp of grapes or infusion of bael fruit, and perhaps a little port wine.

It will be useless to do more than mention other methods of treatment which have been practised. Both opium and morphia have been recommended, in considerable doses; of the former, a grain three times daily; of the latter, a quarter-grain three times a day. Dr. Blecker [1] was the principal champion of this treatment, declaring morphia to be the best of remedies, diminishing the otherwise unbearable pain, moderating the peristaltic action of the bowels, taking away the dryness of the skin, promoting copious perspiration, and procuring wholesome sleep. Opium or morphia judiciously used may certainly prove of great benefit by moderating pain when chloral does not succeed, but I agree with Sir J. Fayrer, who states as a general rule it is well to avoid the use of opiates as much as possible, as they, especially opium, prohibit that which it is an object to produce, viz., free secretion; and although opiates relieve pain, they, especially opium, mask the symptoms of the disease without curing.

All kinds of astringents have been used, such as kino, tannic and gallic acids, compound tincture of benzoin, acetate of lead, nitrate of silver, &c. None of these ever seem to do much good, unless in cases of a hæmorrhagic tendency, which sometimes appear to be lessened by gallic acid; but oil of turpentine, as before mentioned, is the most generally efficacious.

Other agents which have been vaunted as internal remedies are Castile soap in masses the size of a nut,[2] and, stranger still, *vitrum antimonii ceratrum* prepared from the glass of antimony.[3]

[1] *Ind. An. Med. Sci.*, vol. i. [2] *Lancet*, Dec. 29, 1855.
[3] Pringle's *Medical Essays*, vol. i.

Injections of all kinds, large and small, have also been used. Mr. Hare,[1] of the Bengal Service, reported great success in treating dysentery by large injections of warm water pumped into the bowels till the patient complained of distension, the fluid acting as an internal fomentation to the part. But, theoretically, as we know a flow of water will only clean, not cure, an external ulcer, there is no valid reason why it should cure an internal one; and, practically, the injecting-tube being passed as high as possible, hurt the tender rectum, which was in many cases an insurmountable objection. Astringent injections have also been used, especially nitrate of silver, such as from thirty to forty grains in three pints of water.

Charcoal enemata were at one time much recommended, especially when the stools became more than ordinarily offensive. Although none of these means are recommended, it is mentioned that when tenesmus is urgent, with much pain at the lower part of the rectum, a small enema or suppository of opium affords much relief. In other instances fomentation to the anus and abdomen, or a hot hip-bath may be employed, and especially the latter if there is strangury or pain in making urine.

Whatever treatment is adopted, fluid diet and the recumbent posture must be insisted upon.

Chronic Dysentery may commence insidiously without any previous acute attack, but most frequently it results from the acute form, after which soreness remains in some part of the abdomen. Sometimes this may be covered by the point of the finger, at others it occupies a much larger space, while the stools are occasionally slimy and bloody, alternating with constipation for a day or so. The pathological condition consists of thickening and induration with more or less loss of structure, and in many cases an unhealed ulcer or ulcers. If the stools are occasionally bloody

[1] *Ind. An. Med. Sci.*, vol. ii.

or muco-purulent, the probability of the latter is great. Chronic dysentery is also frequently associated with hæmorrhoids, and in insidious cases appears often to commence from hæmorrhoids. When blood and mucus follow a discharge of fæcal matter the hæmorrhoidal condition is indicated, but it is often difficult to diagnose how much of the distress is to be attributed to the one condition and how much to the other. There is also often in cases of chronic dysentery a considerable amount of spasmodic rectal stricture, or even of permanent narrowing of the gut from the loss of substance or the cicatrisation of ulcers. The constant pain and the discharge eventually produce much debility, and the patient is liable from slight causes, as exposure or inadvertence in diet, to attacks of a semi-acute nature, which leave the system still more weak and debilitated, and aggravate the original diseased condition, until the dyscrasia is so pronounced that the material exuded is incapable of healing the ulcers.

Under such circumstances, when there is pain referred to any part of the abdomen, the application of repeated counter-irritants is advisable, the best of which is iodine paint or mustard leaves. If there is constipation the bowels must be regulated by small doses of castor oil, or by sulphur, or by Hunyadi Janos water, which suits some cases best. When the bowels are not constipated astringent medicines of various descriptions may be employed, such as acetate of lead or sulphate of copper in combination with hyoscyamus extract. A mixture composed of compound tincture of benzoin three drachms, compound tincture of catechu six drachms, tincture of opium fifteen minims, extract of logwood one drachm, in six ounces of water, is sometimes very beneficial. Dover's powder and quinine three times a day is useful. If there is alternate looseness and constipation, it will probably be better to trust to diet and castor oil when required, and not to take astringents. But in any

case decoctions, or syrup of Indian bael, may always be tried, as the bael possesses astringent, slightly aperient, and, as it is stated, slightly sedative properties. Bael is the *ægle marmelos* of Linnæus, and belongs to the *Aurantiacæ*, the native name being 'bael geerie.' The decoction is made by boiling three ounces of the dried fruit, or, if obtainable, one ounce and a half of the half-ripe fruit, discarding rinds and seeds, in a pint of water until it evaporates to half a pint, the dose for an adult being a wine-glassful three or four times daily. The syrup is prepared by adding a wine-glassful of water and a teaspoonful of sugar to the soft juicy part of half a moderate-sized bael, rejecting all stringy pieces, and this may be taken three times a day; or, the bael not proving efficacious, decoction of pomegranate may be used, made either with milk or water. These remedies may be frequently changed, as astringents employed in chronic dysentery appear to act most beneficially during the first few days they are taken. Sometimes when the disease appears located in the rectum local medications of the gut may be advisable, and a solution of nitrate of silver, two grains to the ounce, may be used. Dr. McLean's favourite remedy for men returning weak and anæmic and with chronic dysentery from tropical climates is the solution of pernitrate of iron.

But the cardinal points of the treatment are rest, and diet, which should consist chiefly of soup, broth, rice, jelly, sago, arrowroot, or flour and milk well boiled together and seasoned with sugar and spice. As convalescence advances a little well-cooked fowl or flesh may be allowed; generally a little port wine may be given. A flannel should be worn over the bowels, and the feet kept warm by woollen socks. It cannot be too much impressed on the patient that oversight or neglect regarding diet or exposure will negative all medical treatment. During the progress of chronic dysentery indications of scurvy should be duly sought for, and if

the patient has been in a locality where fresh vegetables were scarce, he should have lime-juice, or pulp of ripe grapes, or other antiscorbutics, even although no indications of scurvy are apparent.

Many cases of chronic dysentery are, however, little benefited by medicine, and resist all treatment. In such cases a *thorough change of climate* to Europe is necessary. But the questions of removal of Anglo-Indians and of change of climate are not sufficiently studied. Rest is necessary for the treatment of chronic as well as acute dysentery, and experience should have taught that it is unsafe to send persons on a voyage if there is anything approaching even semi-acute dysentery; for the sufferings of an invalid, especially with dysentery, on board ship are manifold and continuous, without the separate cabins, extra attendance and good sick-cookery which is required, and which cannot be obtained, but which should be obtainable on board all our passenger ships. But in these days of quick transit, under the idea that a change home is all that is necessary, the tropical invalid too often rushes into the cooler climate of Europe, or of the British isles, the sudden change being in most instances as likely to do harm as good. A person in rude health, or with perhaps little the matter, may pass from the tropics to the temperate zone without injury, and often find benefit and enjoy the change. But the tropical invalid, especially if his malady is of long standing, or if he is organically diseased, cannot endure these sudden changes with impunity; and although it is often right that as a last resort they should be tried, they can only be attempted with a chance of success under the greatest care as regards regimen and protection from cold. For the vital energy is often brought so low that the system cannot accommodate itself to the alteration of climate, and the constitution may be so debilitated by tropical heat that return to a cold climate may cause a renewal of, or even

excite morbid activity in, an organ previously diseased or disposed to disease. It is doubly injudicious for a person suffering under any predisposition to organic disease to return home in the winter. Invalids, especially with dysentery, should not reach England till after the vernal equinox, for the gales at that time are often bitterly cold, and are apt to induce congestion of internal organs.

If a change to Europe cannot be accomplished, a change from the interior to the sea coasts is advisable, but not to a hill station, where the lower temperature would probably be injurious. And if benefit does not follow this measure, or if the case commenced on or near the coasts, a change in Western India at least, to the tableland of the Deccan, may be taken. As the lesser of two evils, if the radical change of climate to Europe cannot be taken, a long sea-voyage to Australia, if possible, may be recommended, or otherwise a shorter distance; but, as a rule, voyaging in the Indian seas is not likely to benefit a person with confirmed chronic dysentery. When it is considered that a person transported to Europe may be years before recovery, and that he may be subject to aggravation from the slightest imprudence in hygiene or diet, it is evident that sea-voyages in the tropics are not calculated to cure the malady.

As so many persons require change of climate for chronic dysentery, this seems a suitable place to remark on the cases which *should or should not be sent to the Indian hill ranges*. Convalescents from first attacks of acute dysentery or acute hepatitis, who are *quite* free from structural disease, but who are so debilitated as to render it improbable that they will recover under ordinary circumstances on the plains; persons debilitated by long residence in India, unaffected by any marked disease; or persons suffering from dyspepsia, irregular action of the bowels, or symptoms of functional derangement of the viscera; or young men of weakly constitution, having no acute disease, but who, from debility or

exhaustion, are unfit for duty: all these classes may be sent, with benefit, to the hills—the class first mentioned with the greatest benefit to the intertropical hills, the classes last particularised to the Himalayan stations.

Persons who have been long subject to returns of intermittent, those who have suffered from repeated attacks of hepatitis, or who have long ailed from chronic hepatitis or dysenteric affections, or those who labour under any cardiac or thoracic ailment, or who suffer from rheumatism or from syphilitic or pseudo-syphilitic cachexia, should not be sent to any hill station.

CHAPTER XIV.

DYSPEPSIA.

Dyspepsia, or indigestion, has attained the dignity of being described as if it were a specific disease, instead of a varying combination of symptoms of one or more functional or organic errors of one or more organs; more frequently than not induced by (*a*) deficiency of food, (*b*) excess of food, (*c*) improper food. It is, however, difficult, and frequently impossible, to determine not only what these errors may be, but also what organ or organs are implicated, for there may be both irregularities of the mechanism of digestion (due to such causes as paralysis, spasm, obstruction, dilatation, adhesion, tumours, &c.) and imperfections in the chemical changes (due to such causes as perverted nervous influence, abnormal blood-supply, degeneration of secreting organs, &c.). Similarly it is difficult, and often impossible, to determine what particular errors of diet may be at the root of a dyspepsia. Dyspepsia may therefore depend on *direct* exciting causes, which immediately affect the stomach or other organs concerned in digestion; or on *indirect* causes which influence the digestive organs through the medium of other parts, and of each of these causes there are many. The forms of dyspepsia mostly prevalent in India may be conveniently described under the following heads:—1. Accidental dyspepsia; 2. Hepatic dyspepsia; 3. Gastric dyspepsia; 4. Intestinal dyspepsia (including *atonic*, *flatulent*,

nervous, and dyspepsia *from accumulation in the large bowels.*

1. Accidental Dyspepsia may be of the most trivial character, or it may culminate in a severe so-called bilious attack, preceded or attended by giddiness, faintness, or orbital pain. It may depend on some irritating material taken into the stomach; or on some external cause, as exposure to cold and chill, or great and unaccustomed exertion, as shaking in a rough vehicle immediately after food, interfering with the digestion of ordinary diet. As often as not it may be traced to unaccustomed diet or indigestible food, as salted meats, pork, salmon, rich gravies, high game; or to the combination of indigestible articles so frequently served at a large dinner. Tinned provisions are a fertile source of accidental dyspepsia, not only from the comparative primary indigestibility of many of the viands which are so preserved, but also from the unavoidable increase of indigestibility acquired by the process of preserving; and also from old tinned provisions becoming impregnated with the metal in which they are cased, or with the solder used to seal the cases. *En passant*, it may be observed it is advisable that all preserved provisions for tropical climates should be packed in glass cases. Fish, again, are a frequent cause of accidental dyspepsia, some varieties at particular seasons being especially liable to become tainted or even poisonous, although the taint may scarcely be detected when the fish is served up to table. The evil results of partaking of apparently good oysters or prawns—from slight epigastric uneasiness, nausea, or diarrhœa, to attacks of cholic or choleroid—are frequently noticed in Bombay. Lastly, dirty or improperly prepared cooking utensils may excite similar disturbances. The utensils used in India are of copper, and when properly lined with tin are quite harmless. But the tin wears off quickly, and, exposing the copper, may lead to copper-poisoning; the symptoms of which are

usually pain in the bowels and diarrhœa, and if much copper is taken into the system, also vomiting. If the cooking pots are not properly clean as well as not properly tinned, the probability of copper-poisoning will be increased from the more rapid formation of verdigris by the action of the acids and fats of the food remaining in contact with the metal. Lead-poisoning may also occur as a result of mixture of lead with the tin coating. This, however, is not so likely to happen as copper-poisoning, but the possibility should be borne in mind.

In its milder manifestations, accidental dyspepsia is symptomised by slight eructation after meals, by slight headache, and perhaps a little diarrhœa the following morning, when all is well again. But the attack may be accompanied by giddiness, faintness, nausea, violent vomiting, first of the contents of the stomach and then of sour bilious material, with constipation in the first instance succeeded by desire to stool. Sometimes there is an efflorescence of nettle-rash. Sometimes there is real colic, and this usually comes on suddenly with spasmodic griping and twisting pains in the bowels, and perhaps retraction of the navel. The bowels are usually distended by flatus, the passage of which is attended with relief. This accidental colic arising from ingesta, should be distinguished from the form afterwards described (p. 223) as the sudden attack occurring from acidity. Such an attack usually passes off quickly, but it may be the first step of inflammatory colic or of 'iliac passion' or obstruction. Instead of the patient continuing to find relief from pressure, the bowels may become tender, especially about the cæcum, with increase of temperature and constipation. But accidental dyspepsia, instead of presenting any of the symptoms yet noted, may cause stomach or sick headache. The pain is usually felt in the forehead, or over and about one eye, and is of a throbbing or bursting character, and much more diffused than the more localised, acute, and paroxysmal pain

of true neuralgia; although in persons who have suffered from the latter, accidental dyspepsia will frequently re-excite the neuralgic affection, to the increased suffering of the patient. Sick headache may be attended with thirst, feverishness, and nausea, and these symptoms are most common in persons leading sedentary lives. Pain without nausea occurs to stronger persons, who have exceeded either in eating or drinking, and especially when bad wine or spirits, or indigestible articles of food, have been taken. The pain may last for a few minutes, or for many hours, and generally comes on suddenly after meals, or is felt early in the morning. Sometimes the headache is preceded by the appearance of a small dusky spot before one or both eyes, which spreads as a zigzag halo of light, leaving vision free in the centre, and disappearing after a variable time at the circumference of the field of vision. Occasionally this occurs and is not followed by pain. Sick headache, when the pain occurs over the eye, is often erroneously regarded as neuralgic or malarious, and treated by quinine, whereas the remedies for dyspepsia are required. I believe all these forms of accidental dyspepsia are comparatively more frequent among Europeans and Parsees than natives of other classes, who suffer more from other varieties of dyspepsia.

Treatment.—For occasional minor attacks as first described, no treatment is required. When there is giddiness, nausea or vomiting, or sick headache, especially if shortly after a meal, an emetic of warm water, or of mustard and water will often afford relief. When there is colic as well, after the emetic, a dose of calomel and opium, followed in two or three hours by a saline aperient, are indicated. Pain in the bowels may be much relieved by pressure with the hands, by hot fomentations, by friction with soap liniment, or by a mustard leaf or poultice. If very violent, fifteen or twenty grains of chloral may be given. For sick headache alone, twenty drops of sal-volatile in an effervescing draught

of citrate of magnesia, and a cup of strong tea or coffee half-an-hour afterwards; rest, quiet, and if possible sleep.

2. Hepatic Dyspepsia.—Although in Europe dyspepsia is very often credited to hepatic derangements when a result of other causes, in India dyspepsia more frequently depends mainly on hepatic derangement than on any other cause. Almost every symptom afterwards detailed may arise from some disorder of the liver. Thus acid dyspepsia accompanied by eructation after meals may arise from a deficient secretion of bile retarding digestion, and a so-called bilious attack may depend on overflow of bile into the intestines. Absence of bile by irritating fermentative changes may induce intestinal dyspepsia with all its symptoms, while the elements of bile in the blood may induce that atonic nervous condition so favourable to dyspeptic derangements.

Then there are disorders of the metabolic function of the liver, the principal result being the non-conversion of nitrogenous matters into urea, and the production of lithates and lithic acid inducing the condition designated by Murchison as *lithæmia*. This may be relieved for a time by elimination by the kidneys, showing itself by deposit of lithates or lithic acid in the urine, but afterwards more or less distressing symptoms arise. Such are flatulence, heartburn, furred tongue, viscid mucus in the mouth, with a bad taste especially in the morning, palpitation, irregular pulse, irritability of temper. There is also tendency to gout, urinary and biliary calculi, and diseases of the skin. Individuals subject to *lithæmia* are also more liable to local inflammations which are often regarded as 'gouty.'

Unless in exceptional cases, the treatment of dyspepsia in India, while carried on as noted under the different headings, should still be persistently directed towards securing a healthy action of the liver, by avoiding spirits, starches, and sugars (p. 231), by securing a proper amount of exercise (p. 220), by remedies calculated to maintain the flow of bile,

and consequently the discharge of bile-acids (p. 221), by the removal of *lithæmia*, and by the combat of those various causes giving rise to obstruction of the portal circulation, and the consequent obstruction of the whole of the digestive system.

3. Gastric Dyspepsia.—It seems probable the first step towards gastric dyspepsia is a diminution of the normal alkalinity of the blood, or an increase of acidity in that fluid. Acids of various kinds not only enter the system with food, but are formed in the intestinal canal, and are moreover believed (from the experiments of Gaskell) to be generated in the tissues by the contraction of muscles, lactic acid especially being a product of tissue-metamorphosis. How much acid thus impregnates the system cannot be determined, but it must be considerable, as we know that a large amount is discharged by the lungs, in the gastric juice, by the urine, by the skin, by the liver, and by the intestines. The quantity of acid exhaled by the lungs of a healthy man has been shown by Dr. E. Smith to average about 950 grammes in twenty-four hours. But it is affected by external influences, being for instance increased by exercise and lessened during repose and sleep. The atmosphere of the tropics is from the heat more rarefied than that of a temperate climate, the result being that a given bulk of inspired air must contain less oxygen in the former climate than in the latter. And as very generally comparatively less exercise is taken in the tropics, and often more sleep (owing to the small amount of cool, suitable time available, and to the lassitude induced by heat), it follows that the breathing is less accelerated by motion, resulting in a diminished bulk of air being inspired, and hence again a smaller amount of oxygen. As a necessary consequence of these *two* distinct manners in which the supply of oxygen is curtailed, the carbon breathed out from the lungs in the shape of carbonic acid is diminished. In other words it may be stated, the lighter the temperature and the less the exercise, the less carbonic acid is exhaled from the lungs.

As regards sedentary habits leading to the accumulation of acid in the system, Parkes showed that in a temperate climate more oxygen is taken in and more carbonic acid evolved on a working day, to the extent of one and a half ounces. On the other hand, from over-fatigue or exhaustion the blood circulates less vigorously, removal becomes slacker, and the products of combustion accumulate in the system. The acid, chiefly *acid sodium phosphate*, which passes off by the urine, is also, it is believed, diminished in quantity in proportion as the secretion and flow of urine is itself diminished in warm latitudes. But at present we have no reliable experiments on this point. By the skin, six grammes of carbonic acid are exhaled in twenty-four hours, also an undetermined quantity of formic, acetic, butyric, and other acids in the sweat. And although it is true perspiration is more copious in a tropical climate, it does not follow that there is a greater discharge of acid; but, as with the acidity of the urine, we are without reliable experimental observation. We know, however, that all acids passed by the skin are increased by exercise, which, as above stated, is usually less freely taken in a tropical atmosphere. By the liver, in combination with alkalies, at least two acids are discharged. Lastly, there is a considerable formation and discharge of acid from the intestines.

By these various methods the acids introduced or formed within the system being continually removed, the blood has always an alkaline reaction. So long as the discharge of acid passes off, and is distributed in a natural manner with the excretions, its presence on the mucous surfaces or in the various organs is unrecognised. But when acid is in excess, the result of incomplete oxidation of the elements of the tissues and of the food, or the result of the deficient elimination of acid formed in normal or abnormal quantities, it may be elaborated and passed off by various organs. Then secondary effects due to acidity present, or as more scientifi-

cally stated by Ralfe,[1] 'a physiological check occurring to the elimination of matters by their natural passages, causes an outbreak in another direction.' Gastric or acid dyspepsia is not to be regarded, then, as accidental dyspepsia, the consequence of undigested food, but as the result of a continued course of high and improper living, which, taken for some time with seeming impunity, at length induces (especially in a tropical climate) that condition to which the term bilious is so commonly applied. But the first step appears to be hypersecretion of gastric juice. We know that gastric juice is mostly, if not entirely, secreted when a stimulus is applied to the stomach, but this stimulus need not be food. It is believed that a departure from the normal alkalinity of the blood may lead to either direct or reflex irritation of the nerve-centres of the mucous coat of the stomach, and so to hypersecretion of gastric juice. On the other hand, many forms of nervous disturbance have a powerful influence in retarding the secretion of the gastric fluid, as grief, fear, anxiety, overwork, fasting, hysteria, thus leading to the accumulation of acid elsewhere, and eventually to reflex action and hypersecretion.

When the formation of acid in the stomach is only slight, or temporarily in excess, the symptoms may by limited to slight heartburn, to a little acid rising, to irritability of the urinary passages, or to nettle-rash. Or it may be manifested by irregularity of the pulse, palpitation or intermittent action of the heart, aching of the limbs, noises in the ears, vertigo, depression, insomnia, head-ache, erythematous patches, and other anomalous or ill-defined symptoms. When the acidity is more confirmed or long continued, there may be pain of a gnawing, burning character, relieved by taking food. There may also be vomiting of tough mucus on an empty stomach, or provoked by taking food, although these last symptoms do not occur at first, and are most

[1] *Morbid Conditions of the Urine*, 1882.

usually found in persons habitually taking alcohol in excess. In this acid form of gastric dyspepsia, the urine is usually loaded with lithates.

But acid dyspepsia may culminate in a severe so-called bilious attack. Thus a person after probably complaining of frontal headache, begins to suffer from uneasiness and then pain in the epigastrium, shortly afterwards vomiting and continuing to do so for some hours, bringing up first a glairy acid fluid becoming more and more tinged with bile, till apparently bile only is discharged. The attack then passes off, leaving some depression, and if there are frequent attacks, a pallor which may be mistaken for the sallowness of bile. In such a case the bile does not proceed from excess of secretion, but from the emptying of the gall-bladder by the action of vomiting, and the liver is not therefore immediately in fault. In these instances, although the urine may be acid at first, it eventually becomes alkaline, remaining so for an indefinite period, probably from the withdrawal of acid from the system by the violence of the attack.

A still more serious demonstration of gastric acid dyspepsia is the condition known as *gastric fever*, which in its most aggravated form is apparently the malady styled by French physicians *la dyspepsie acide grave*. By gastric fever is meant the state of the stomach arising from improper food inducing undue acidity in the system, and not the irritable condition of the stomach arising during, and caused by, fevers and other exhausting diseases, and to which the term *gastric fever* is often wrongly applied. When the stomach is disordered by acidity and hypersecretion of gastric juice, the attack may not pass off as previously noted in the shape of headache, or skin-eruption, or vomiting, or a so-called bilious attack. There may be increasing pain and tenderness at the pit of the stomach, with constant hiccough, nausea, and vomiting, even water being rejected. The vomited material consists of saliva often tinged with bile,

and there is intense sourness of the breath, so that the surrounding air is tainted. But a disordered stomach, even when attended by the acute symptoms above detailed, will usually recover itself in the course of a few days. If the symptoms continue for a longer period, there is every reason to fear that some form of fever, or in children hydrocephalus, may be the cause of the gastric disturbance. It is in the diagnosis of such cases that the history of the patient is so useful. If the stomach symptoms came on after indulgence or improper food, and if there is no typhoid in the neighbourhood, it may be safely concluded the ailment is not typhoid. But it may be so-called remittent, and this is more likely if there has been no prior cause for disordered stomach, or if the person has been exposed to the sun, or sleeping in damp malarious localities.

Attacks commencing with all the symptoms of acid gastric dyspepsia, when culminating as described in so-called gastric fever, have been noted as terminating after many days in nervous symptoms or coma. I doubt if these are true attacks of gastric dyspepsia. I believe such cases are hydrocephalus, whether in children or adults, and that the gastric symptoms are secondary to the irritation of the brain.

Among the more remote consequences of acid gastric dyspepsia may be mentioned inveterate skin-diseases, asthmatic paroxysms in those liable to asthma, gout when this hereditary diathesis prevails, and gravel. It is indeed the mucous membranes and skin which chiefly suffer as the result of prolonged chronic acid dyspepsia. The former become subject to catarrh from the irritating presence of the acid. The same result occurs in the skin, while abnormal acidity of urine produces not only a catarrhal stage of the urinary passages, but, by decomposing the salts of uric acid, causes a deposit of insoluble uric acid, thus giving rise to attacks of gravel or to calculus.

The *treatment* of acid dyspepsia is chiefly by dieting and

hygiene. The habits of natives are very provocative both of this and other forms of dyspepsia. Many of them distend the stomach once daily with rice and dall, or in the northerly districts with bajree and dall; at times they consume large quantities of sweetmeats, and they frequently after eating drink very large quantities of water. Natives suffering from dyspepsia should substitute wheat flour for much of the rice they consume, they should abandon sweetmeats, the stomach should not be distended with fluid, food should be taken more frequently than once or twice daily, and a little early in the morning. In the case of Europeans the diet should be nutritious, but easy of digestion, and the meals should neither succeed too rapidly nor be too long deferred. Five hours at most should elapse between meals, and the stomach should never be overloaded. A little food may advantageously be taken the last thing at night. Both alcohol and coffee are to be avoided, also tobacco. When heartburn or acid risings are the prominent symptoms, bi-carbonate of soda or prepared chalk will often give temporary relief, or compressed soda-mint tablets may be used. When an intermittent pulse and the anomalous symptoms mentioned at p. 222 present, an occasional aperient, and very careful living are indicated; pain may be relieved by five minims of *liquor opii* or *liquor morphiæ* with a few grains of bismuth, or by chloral. Vomiting may be relieved by the above, or by a mixture containing dilute hydrocyanic acid thirty minims, spt. chlorof. two drachms, tinct. of ginger two drachms, aquæ eight ounces; or drop doses of ipecac tincture may be given every hour; or drop doses of *Liquor arsenitis potassæ* may be tried, with such local measures as a mustard leaf or wet compress to the epigastrium. Should acid dyspepsia culminate in a so-called bilious attack, the treatment indicated at p. 218 may be pursued, but in most cases it is as well to let the patient alone so far as energetic medical treatment is concerned.

The stomach disorders of infants and children, being mainly acid dyspepsia, may be mentioned here. Gastric disorder is more common in infants being brought up by hand than in those who are suckled. It may be caused by very slight uncleanliness of the feeding-bottle, especially about a cork, in the crevices of which decomposing milk may lurk, and so contaminate successive contents of the bottle. Or it may be caused by improper food, or by over-feeding. Very sour breath, vomiting after food, flinching from slight pressure at the epigastrium, flatulence, and fever are characteristics. Children thus affected also usually suffer from diarrhœa, and the stools may be light of colour, and containing many lumps of undigested milk. This acid dyspepsia of children most usually subsides in a few days, but if the cause is not removed, or very potent, it may terminate in one or other of the following conditions: 1. *Thrush* or *aphthæ*, consisting of an eruption on the tongue, lips, interior of the cheeks and gums, of small vesicles which discharge a whitish mucus like morsels of curd, for which they may be mistaken (believed to consist of the microscopical vegetable parasitic growth, *oïdium albicans*), and which falling off disclose small ulcers. 2. Infantile *diarrhœa* (*vide* p. 174). 3. *Dysentery*, which is often a sequel of diarrhœa. 4. *Remittent fever*, which in children occurs from a number of causes (of which improper diet is one of the chief), and not from malaria alone. 5. *Convulsions*, of which turning in of the thumbs to the palms of the hand, twitching of the face, starting during sleep, and squinting are the premonitory symptoms. 6. *Hydocephalus spuriosa*, of which the principal symptoms are moaning, whining, starting with a louder cry, eyes half open, a pale cheek, a cool skin, absence of a continued febrile condition, and a sunken or depressed *fontanelle*. 7. *Atrophy*, which at first being simply wasting from absence of a necessary supply of food, eventually, especially in children with a scrofulous tendency, becomes atrophy

from enlargement of the mesenteric glands—an enlargement first excited by malnutrition, and afterwards itself preventing nutrition.

4. Intestinal Dyspepsia.—Dyspepsia may arise not only from an excess of acid in the blood leading to irregular or excessive secretion of gastric juice, but also from fermentative changes taking place in the alimentary canal, which interfere with what should be, in the upper part of the small intestines, the pure alkalinity of the contents emulsionised by the bile and pancreatic secretion. In a healthy condition no fermentation takes place in the stomach, where the gases found are oxygen, nitrogen, and carbonic acid. In the lower half of the small intestines acetic, lactic, and butyric fermentations commence, reaching the acme in the large intestines, where carburetted and sulphuretted hydrogen are found. It is the sugar introduced with the food which furnishes most of the acid gases developed. Acetic acid gas is formed by the fermentation of amylaceous and saccharine material. Rancid gases are formed by the decomposition of nitrogenous matter. But this may occur independently of food by the decomposition of the mucus of the stomach and intestinal canal, which may account for rancid flatulence being present, as it often is when the parts are empty. Fermentative changes may indeed commence in the mouth by decomposition of the oral mucus.

The principal *causes* inducing intestinal dyspepsia depending on fermentative changes within the canal are, generally speaking, those causes which interfere with or retard digestion. Among these are food of indigestible nature, or of bad quality, especially if itself fermenting; excess of food, so that much of it not being absorbed remains in the bowels; eating too quickly, or when excited; too free consumption of amylaceous and saccharine matter, which so easily undergoes acetic and lactic fermentation; indifferent mastication of the food, so that the stomach cannot reduce it

into chyme, too much food at one time; too much liquid, especially tea, the action of the tannin in which on albuminous principles interferes to some extent with their digestion. Moreover, tea is a vehicle for sugar and milk, the former as above noted so readily undergoing acetic fermentation, and the latter containing the elements of acetic acid fermentation. Bile exercises an undoubted anti-fermentative action, and induces increased peristalsis of the intestines. Anything, therefore, interfering with the flow of bile must increase fermentative or intestinal dyspepsia. Thus acidity in the duodenum by creating catarrh interferes with the flow of bile, and thus tends to intestinal dyspepsia. In ordinary dyspepsia of the kind there is weight and fulness after taking food, or tearing, twisting sensations, or colicky pains. There is also rising of undigested food, and of sour fluid which sets the teeth on edge. These sour risings contain lactic and acetic acid, and when rancid as well as sour, butyric acid. When these symptoms are prominent the acid may be sufficiently powerful to coat copper vessels. Micturition is usually frequent, especially at night, and the urine is generally alkaline, often presenting on standing an iridescent film on the surface, and containing oxalate of lime crystals. There are commonly weariness, muscular pains, sometimes dyspepsia, and the conjunctivæ are often yellow.

But intestinal dyspepsia may assume a decided *atonic*, *nervous*, or *flatulent* form. It is stated by Prout that malarious seasons or districts aggravate the tendency towards saccharine mal-assimilation. This, however, most probably results from the vitiated condition of blood inducing that anæmia characteristic of so-called malarious influences. A debilitated state of system must be accounted as holding a foremost place in the production of fermentative dyspepsia of the atonic form, as in such condition the propulsive action of the muscular coats becomes feeble, the onward passage of

food is delayed, the secretions concerned are defective in quantity and quality, and fermentative changes are the result. Mental strain, depressing moral influences, and bad hygiene are usually much concerned. As the dyspepsia assumes an atonic character there is the same weight and discomfort after taking food, often referred to the chest rather than the stomach, and when felt in the bowels of a colicky nature. The bowels also sometimes feel tender with flatulent distension, and there may be uneasiness in the right or left hypochondrium; there are sour risings or regurgitations two or three hours after food. Fluid brought up may contain sarcinæ (a genus of microscopic fungi, belonging to the order saccharomycetes, composed of constituent cells arranged in groups of four, sixteen, or thirty-two), and often indicative of structural change. There is loss of appetite and of flesh, a broad tongue covered with thin white fur, with indented edges, and the heart's action is feeble, perhaps intermittent. The urine is alkaline, with tendency to deposit phosphates. In many instances there is considerable nervous disturbance, the patient being easily excited, alarmed, or depressed. Such disturbance hastens the passage of aliment through the intestinal canal, causing diarrhœa, usually of a yeasty character, and which only temporarily relieves the flatulence and sour eructations. The mischief resulting from the excessive formation of acid in the intestinal canal is not limited to mere disturbance of digestion. It produces injurious effects on the system, both muscular and nervous, by interference with nutrition, and it thus directly tends to induce that state of debility, and that irritable nervous condition under which dyspeptic derangements arise, and thus the malady itself becomes a cause of its own aggravation.

It is atonic dyspepsia which generally assumes the flatulent form, when flatulency is the principal subject of complaint. If the risings are rancid, as they generally are,

fermentation of nitrogenous material is indicated. The enormous quantities of gas passed by hysterical or hypochondriac patients does not appear to be caused by fermentative changes, as it is almost inodorous, chiefly carbonic acid, but it is probably derived by diffusion from the blood. Both atonic and flatulent dyspepsia are common in India among natives, and probably result from weakness through impaired innervation which the digestive organs share in common with all parts of the system.

It is also in atonic dyspepsia that the excessive secretion from the glands of the stomach, known as pyrosis, occurs. This presents most frequently among the poorer classes of northerly natives who live chiefly on 'bajree,' and as with the people of North Scotland, who suffer much from pyrosis, the malady has been attributed to the effects of an irritating or of an innutritious diet. But the cause why the stomach should periodically secrete large quantities of clear alkaline fluid, sufficient to neutralise any acidity, to excite burning sensation at the cardia, or even to excite vomiting, lies deeper than mere diet, and may, perhaps, be an effort of nature, often carried to excess, to neutralise acids formed in or entering the viscus.

The principal features of distinction between gastric and intestinal dyspepsia are as follows:—In gastric dyspepsia there is pain of a gnawing character, relieved by taking food. In intestinal dyspepsia there is a feeling of weight and distension of the stomach, but not actual pain, or griping in the bowels some time after taking food. In gastric dyspepsia there may be vomiting of acid fluid; in intestinal dyspepsia sour risings without vomiting; in gastric dyspepsia little or no flatulence; in intestinal dyspepsia flatulence a more or less marked symptom. In gastric dyspepsia when a so-called bilious attack takes place it resembles colic; in intestinal dyspepsia, although there are colicky pains, a so-called bilious attack rarely occurs, unless

there is both gastric and intestinal, or *gastro-intestinal* dyspepsia.

Although the different forms have been enumerated and described as distinct maladies, although typical cases of each kind frequently occur, and although both have distinctive symptoms, it still more frequently happens that both gastric and intestinal dyspepsia exist together, one or other being prominent alternately, for one may cause the other. Thus hypersecretion of gastric juice, or pure gastric dyspepsia, may itself induce fermentative changes, over-acidity preventing the digestion of albuminous substances. The acid fluid from the stomach containing imperfect products of gastric digestion passing into the intestines excites catarrh, first of the duodenum, and then of lower parts of the passage. In the duodenum it interferes with the discharge of bile by creating a catarrh or irritation of the mucous membrane. Hence persons subject to gastro-intestinal dyspepsia are usually sallow, and more liable to so-called bilious attacks. On the other hand, the irritation produced by fermentative changes may excite hypersecretion of gastric juice in the stomach from, as is presumed, the disturbance of the nerve-centres of the stomach by the irritation excited by the products of fermentative changes in the blood.

In the *treatment of intestinal dyspepsia* the chief objects are to prevent decomposition in the alimentary canal, to secure solutions of albuminous compounds, to lessen the quantity of saccharine matter taken, and to give tone to secure propulsion. As with gastric dyspepsia, dieting is the chief consideration. The food must be nutritious, well cooked, and well masticated, taken frequently—about every four hours—and in moderate quantities. Some light article of food should be taken both before going to rest and on rising in the morning. Food of indifferent quality must be avoided, and saccharine articles of diet reduced to the smallest possible limit. It must never be lost sight of that it is the

sugar taken with food and drink which furnishes most of the acidity developed. As little fluid as possible should be taken at meals, although it may be supplemented, if thirst prevails, by a moderate draught an hour afterwards. Cream should be used instead of milk. The injurious effects of tea have already been mentioned (p. 228). Alcohol should be avoided. If any spirit is taken it should be pure whisky, which contains less sugar than any other liquor. Beers contain much sugar, and should therefore be abandoned. The wines most rich in saccharine matter are port and sherry, and are therefore unadvisable. The same objection applies to burgundy, which, although less rich in sugar than port and sherry, contains more than the lighter wines. Sound claret, although containing a considerable portion of free acid, has less sugar than most other wines, and is therefore generally borne best. Next to dieting, the avoidance of severe mental strain or physical exertion, of depressing moral influences, of bad, general, or personal hygiene, and of so-called malarious influences, are the points to be taken into consideration, especially when the dyspepsia assumes an atonic form. Removal from all such influences and a dry non-malarious locality are required. It is in such cases that quinine in combination with dilute sulphuric acid may often be given with great advantage. When fulness or pain after taking food are the prominent symptoms. a mixture composed of subnitrate of bismuth four drachms, dilute nitric acid three drachms, tincture of nux vomica two drachms, peppermint water or plain water four ounces, may be taken in one- or two drachm- doses immediately after meals; or an alkali before meals, twenty drops of dilute hydrochloric acid after meals, and pepsine with the meals. Capsicum is especially useful in that dyspepsia of long residents in tropical climates which evidences itself chiefly by a sense of fulness and distension after meals, and the following formula may be employed: capsicum three grains, compound rhubarb pill five grains,

ipecac powder one quarter grain, to be taken an hour before dinner. When there is rising of undigested food or sour fluid, two-ounce doses of aqua calcis, or one- to two-grain doses of iodide of potassium, will often prove useful. There are few better remedies for flatulence than peppermint water, or if flatulence is combined with spasmodic pain, sal volatile in camphor mixture. If the flatulence has a sulphuretted hydrogen flavour, charcoal biscuits or a drop of creasote or of carbolic acid on sugar. As a general rule, acids are most suitable in atonic dyspepsia. Hydrochloric acid is best suited for dyspepsia with feebly acid or alkaline urine, unattended by phosphates; phosphoric acid when the urine is alkaline with triple phosphates; nitro-muriatic acid when the secretion is acid with oxalates or urates. Acids may often be advantageously combined in atonic dyspepsia with tincture of nux vomica. Sour milk after meals has been much recommended in atonic dyspesia by a continental physician, who refers the resulting benefit to the lactic acid sour milk contains. When nervous symptoms predominate, small doses of strychnine and phosphorus may be tried; or oxide of zinc may be given in two- or three-grain doses at night. For females with atonic dyspepsia, with loss of appetite, debility, and, as is often the case, some accompanying uterine disorder, ten-grain doses of effervescing citrate of iron may be beneficial, and if a laxative is required, extract of aloes one grain, extract of belladonna and nux vomica each one quarter-grain. As a rule, however, aperient and purgative medicines are better avoided, unless there is decided constipation present, when aloes for weakly persons and Carlsbad salts or Hunyadi Janos water for stronger individuals will be advisable. Recently, *extractum pancreatis* has been much recommended for intestinal dyspepsia. By giving, it is said, five grains of this preparation after the stomach digestion has been mainly accomplished, and the chyme has passed into the intestine, the pancreas ferment

will complete the digestion of starch begun in the mouth, and of albuminous substances which are only partially peptonised in the stomach. But as pancreatine never gets into the stomach naturally, and as it is destroyed by the gastric juice when placed there artificially, experience is required to demonstrate whether or not pancreatic extracts are more useful than they would theoretically appear to be. It is impossible to mention the multitude of remedies which have been advised for the protean varieties of dyspepsia, the foregoing being merely indications of the most desirable. But it may be observed that benefit has resulted in fermentative dyspepsia from the plan advocated by Dr. J. H. Salisbury, of taking about a pint of *hot* water three or four times a day, midway between meals and on an empty stomach. The water should be as hot as can be conveniently taken, and should be sipped or swallowed slowly.

Dyspepsia from accumulation in the large bowels, which has been included under intestinal dyspepsia (p. 216), is considered under constipation (p. 143).

CHAPTER XV.

ELEPHANTIASIS.

Synonyms: *Elephantiasis Arabum, Bucnemia, Barbados leg, Cochin eg, Egyptian Sarcocele, Tropical big-leg, Nævoid Elephantiasis, Varix lymphaticus, Lymph-scrotum.*

ELEPHANTIASIS, whether affecting the limbs or genitals, is the same disease, and if of long standing its effects are not limited to external changes, but disease of internal organs will be found. It may be defined as consisting constitutionally of a peculiar fever recurring at irregular intervals ; and locally of hypertrophy of the skin and subjacent areolar tissue, due to adventitious deposits, accompanied by deterioration of blood, and change of structure in internal organs, particularly the spleen.

Elephantiasis is frequently seen in Western India, but is perhaps more common in Madras and Bengal. In Cochin, Day stated that about one in seventeen of the native Christians are affected, and of the Portuguese about one in eighteen. Waring stated that in Cochin scarcely a house is free. Even in such districts it is more prevalent in low damp situations near the sea-coast, or in the neighbourhood of marshes, or in any place where tainted or malarious atmosphere might be expected. Others have regarded elephantiasis as limited to the rice-producing countries, which are chiefly the tracts near the sea-coasts. Chevers states it always occurs within the range of the sea-breeze, and that it is never developed

up country or beyond the limits of the growth of the cocoa-nut tree. Manson asserts that the geographical distribution of elephantiasis is the same as that of the mosquito. The truth, however, is that, while occurring in the interior, it is more prevalent on the sea-coasts.

The better classes of natives are comparatively free from elephantoid disease, but it is very prevalent among agriculturists exposed to humid air and damp ground in hot districts. It prevails in almost equal degree among Hindoos, Mahomedans, and Parsees. Europeans are rarely affected, which has been attributed to their not being reared in the country and exposed from the days of their youth upwards to subtle malarious agencies; from their being better fed, nourished, and cared for in a sanitary sense than any of the lower classes of natives, among whom the disease principally prevails, and from so many not remaining long in the country, the majority being invalided or dying of some more acute disease before elephantiasis has had time to develop itself. Fayrer states he only saw two cases in Europeans. I never met with one, but recollect that cases have been reported even in England, occurring in persons who have never been abroad; one of the most recent being brought before the Medical Society of London by Dr. Heath Strange, when Mr. Bryant mentioned he knew a case in Leicester, where the disease had been apparently transmitted through three generations, none of the members of the family having left the town. But Sir J. Fayrer did not consider the cases met with in England as examples of true tropical elephantiasis, which begins with febrile disturbance and rapid hyperplasia, followed by slower growth.

Males appear more subject to the disease than females, in the proportion, according to the combined statistics of Waring, Sheriff, and Richards, of sixty men and forty women in every 100. It may occur both in childhood and old age. Webb saw a sucking child affected. It is, however, rare before

fifteen, and the period between thirty-five to forty is the time when most suffer. It is seldom met with after fifty. Quadrupeds and birds are occasionally attacked.

Symptoms.—The parts affected locally are, as regards, frequency, the lower extremities (usually one, sometimes both) the scrotum, the penis (sometimes the prepuce only) the *labiæ pudendi*, the *mammæ*, the upper extremities, and lastly the face. The developed attack consists of constitutional and local symptoms, but the former may endure for a length of time without evidence of the latter ; or the local symptoms may present without any marked constitutional disturbance. In some patients there is very little constitutional disturbance at any time, and febrile paroxysms are few and far between. The pain is sometimes insignificant, the patients complaining only of inconvenience and discomfort from the weight of the parts affected. In other cases, the pain is intense, the fever recurring at short intervals and quickly exhausting the patient. Occasionally the growth, having attained some bulk, remains quiescent, and constitutional symptoms abate or cease altogether. In typical cases the disease is ushered in by well-recognised febrile symptoms, which are known as *elephantoid fever*. This fever is often marked by the well-known cold, hot, and sweating stages, at other times it is less paroxysmal. It often occurs monthly, and Goodeve and Wise thought it influenced by the moon, while Webb called it 'moon fever.' Quite recently, Chattajee speaks of the regular monthly exacerbation ; but the fact is, the fever may and does usually occur more frequently than monthly, although when it occurs with the phases of the moon it is better recollected. During the paroxysms of fever, there is often vomiting, and severe lumbar pain, sometimes extending into the groin and to the spermatic cord and testes. Occipital pain has also been frequently noticed as an accompaniment of the fever. An enlarged painful gland, becoming more prominent during the fever, usually exists in some

portion of the affected limb, between the site of the following effusion and the patient's body. Thus, when the limbs are affected, the enlarged gland may be in the flexure of the knee or arm, or, as more frequently occurs, in the axilla or groin. Then a dusky line often forms in the course of the absorbents, succeeded after an interval by redness, heat, and swelling of the surrounding parts, with tenderness and enlargement of the neighbouring lymphatics and veins. This subsides in a few days, leaving some amount of thickening and hardness. At this period the dusky line, now with cord-like feel, may still be generally perceived along the course of the absorbents, between the local effusion and the enlarged glands. At irregular intervals the febrile condition recurs with attendant hyperæmic action in the parts locally affected. Although the constitutional symptoms may vary in degree, and although the local swelling may alternately enlarge with and decrease after the constitutional manifestation, the progress of the local affection is nevertheless onwards, until it attains an enormous weight and size. During the progress of the disease, one or more small punctiform openings may appear on the affected part, or above the affected part, from which a milky fluid may be emitted. The parts affected are hard; at first there is superficial redness, afterwards the surface of the skin becomes rugose, and if the leg is affected it resembles an elephant's leg, the feet and toes being more or less hidden by the growth.

Although elephantiasis often commences as above, it may originate in a different manner under the guise of the condition known as *varix lymphaticus*, *nævoid elephantiasis*, and *lymph-scrotum*; the latter not being so applicable a term because the conditions described below may also present on the leg as the forerunner of elephantiasis; thus, as Fox and Farquhar state, apparently proving elephantiasis of the leg and scrotum to be identical with varix lymphaticus,

giving out a chylous discharge. Though many cases have been published, particularly by Lebert, of lymph discharge from different parts of the body, especially from the legs and groins, and although lymph discharge has been alluded to in descriptions of elephantiasis by various authors, its recognition as a distinct pathological condition dates from 1854 by Mr. Ardaseer Jamsetjee.[1] Dr. Wong of Canton then recognised it, in 1858.[2] The next most important note was by V. Carter[3] in 1861. In 1866 Fayrer[4] termed it nævoid elephantiasis, since then McLeod[5] has described it as varix lymphaticus, and still later it has been investigated by others, especially by Lewis[6] and Manson,[7] who states the description of the ordinary commencement of elephantiasis is that of lymph-scrotum.

Nævoid Elephantiasis usually initiates with ague or fever, accompanied by swelling of the inguinal or femoral glands, which sometimes show an erysipelatoid blush. Then, either with or without such fever being recognised, the characteristic feature is the formation on the surface of the scrotum of dilated or varicose lymphatic vessels, followed by the appearance of tubercles or vesicles, which when they rupture spontaneously, or are pricked, discharge a coagulable lymph. Some have described minute orifices on these vesicles, but such orifices do not show at first. The number and size of these varices differ much; there may be two or three or a hundred, and they may be as small as a millet-seed or as large as the tip of the finger. The quantity and character of the discharge also varies from drachms to pounds, from clear white or straw-coloured to salmon colour, or to the colour of blood, often varying as it flows. These variations of hue depend on the lymph discharged being more or less

[1] *Trans. Bombay Med. and Phy. Soc.*, vol. ii. [2] *Ed. Med. Jour.*, 1860.
[3] *Trans. Bombay Med. and Phy. Soc.*, vol. vii.
[4] *Clinical Surgery in India.* [5] *Ind. Med. Gaz.*, Aug. 1874.
[6] *Report of San. Com. with the Govt. of India.* [7] *Lancet*, 1884.

advanced in its transformation into blood. Under the microscope corpuscles like those of the blood are found in abundance, and in many instances active embryo filaria. If the inguinal and femoral glands are enlarged, it has been proved by tapping that they contain fluid having similar microscopical appearances. Sometimes enlargement and a corrugated appearance of the scrotum precede these vesicles, but in the majority of instances follow them. The case may remain as nævoid elephantiasis for an indefinite period, or it may assume more of the characters of elephantoid growth. Chylous urine is also occasionally an accompaniment, in which cases Carter opines that dilated lymphatics exist on some part of the mucous membrane of the urinary tract, as well as on the scrotal integument (*vide* p. 140).

The *surface* of an elephantoid swelling becomes rough, reddened, or livid, or of a dark Indian ink colour, often cracked or fissured, and exhaling a fœtid serous or bloody discharge. Sometimes there are scaly excrescences resembling psoriasis, ulcerated spots, or soft fungoid granulations, while in almost every case enlarged veins traverse the surface. As the disease advances, suppuration, both superficial and deep-seated, occurs, and in a few instances sloughing, from which the patient sinks exhausted after a longer or shorter period. Sometimes death takes place suddenly from embolism or fatty heart. An elephantoid scrotal tumour has been known to weigh 180 lbs., and may be complicated with large hydrocele. The average size of the leg at the ankle is twelve inches, and at its greatest circumference fifteen inches, but it has attained the size of thirty-six inches (Richards). In exceptional cases the enlargement of the leg extends to the thighs, or even to the body (Day).

The *post-mortem appearances* have added little to our knowledge of the pathology or treatment of the disease. Fayrer regards the enlargement as simple hypertrophy of the tissue-elements, progressing most rapidly where lymphatics

abound. The epidermis and cutis generally are found much thickened, and owing to such thickening the ducts of the sweat-glands are elongated. The thickened parts appear to consist of fibro-cellular tissue, interlaced with strong shining bands containing also more or less fat-globules, and an immense quantity of clear serum. The capillaries are dilated, thin, and full of blood. The lymphatics are dilated into irregular spaces, and the glands are in a state of chronic inflammation. Arteries and veins, especially the femoral, when the disease is in the extremity, are also inflamed. The nerves are usually found enlarged, the posterior tibial to the size of the sciatic, and the latter to one-third larger than normal. Softening, enlargement, and amyloid degeneration of the mesenteric glands and of the spleen are frequently observed; also an amyloid condition of the kidneys, with fatty liver and heart. Micrococci, probably connected with the septic process, have been found in the vessels in the vicinity of ulcers.

Causes.—The Hindu, Greek, and Arabian authors assigned its production to tainted air, unwholesome diet, and when attacking the genitals to impure sexual intercourse, which, according to Webb,[1] is still a frequent cause of the latter form of the disease. The same author stated there are two varieties, one syphilitic, and either hereditary or not, the other malarious and connected with 'moon fever,' and that the majority of the cases affecting the scrotum are syphilitic. That syphilis, either hereditary or acquired, is a powerful agent in inducing predisposing cachexia cannot be doubted, but that syphilis and any form of elephantiasis are the same malady is not a generally accepted view, although, as in a case operated on by Wiblin,[2] it may follow syphilis. Malaria of course has been credited as the cause, under the argument that the disease usually occurs in so-called malarious localities and that the elephantoid fever is distinctly paroxysmal;

[1] *Ind. An. Med. Sci.*, vol. iv. [2] *Lancet*, Nov., 1862.

that it is often observed prior to the local swelling; that anti-periodics are often efficacious remedies (which is questionable); that spleen disease is frequently found associated; that splenic leucocythæmia frequently co-exists; and that, as Waring observed, 'the rule is no fever no disease.' Most writers twenty years ago, as Waring, Leslie, Hillary, Musgrave, Day, held these views. Peet, however, regarded the fever as nothing more than sympathetic constitutional disturbance, and not of malarious origin; and Wise asserted the fever to be the result of the local affection, and that the latter is the result of inflammation of the veins. Morehead observed that the use of fermented toddy was favourable to its production, just as wine and beer are to that of gout; and many have thought that different kinds of diet have an influence in producing both this disease and leprosy, in the same manner as 'kessaree dal' grain has been proved to cause loss of power in the lower extremities, or even perfect paraplegia,[1] in those obliged by the *res angustæ domi* to live on this material; or as diseased rye is known to cause mortification. Its prevalence in the rice-producing districts led to the idea that the use of an inferior description of, or of diseased rice, or of a too general rice diet, has an effect in producing elephantiasis, and in proof of this it was advanced that an inhabitant of Bengal will very likely lose the disease if he goes to the provinces of Upper India (a result which by no means follows); the diet of the inhabitants of the latter being more cereals than rice. Others have regarded a fish diet as productive of the disease. But some, as Gould, speak of many fishermen being attacked; others, as Richards, notice a small proportion of fishermen suffering; the explanation of such discrepancy being probably, as Fox and Farquhar suggest, the varying dampness of soil, humidity or air, and heat, where the different observations were made.

Others, who have not been able to reconcile elephantiasis

[1] Irving, *Ind. An. Med. Sci.*, vol. xii.

as excited by any one special cause, have fallen back on generalisation, attributing it to those influences which deteriorate the blood and establish cachexia, such as bad water, hard work, want of rest, deficiency of food, exposure to cold and damp, neglect of sanitary precautions, and the scorbutic diathesis.

The most recent theory of the causation of elephantiasis is that which attributes it to embolism of the lymphatics excited by the ova of filaria. In 1871 Dr. T. Lewis discovered filaria in the milky fluid from a lymph-scrotum, and then suggested they might give rise to obstruction in lymphatic vessels, and to the disease. In 1872 Lewis[1] also found filaria (*filaria sanguinis hominis*) in the blood. The worm is found in this fluid as an attenuated threadlike worm, of a whitish colour, with smooth cuticle or sheath, devoid of transverse marking, but possessing an oval and anal aperture. The head is club-shaped, without boring apparatus, giving rise to the suspicion that its home must be in the blood or lymph. The diameter is about $\frac{1}{3000}$ of an inch by $\frac{1}{68}$. It belongs to the genus *Nematoda*, which includes many other entozoa, as ascaris, dracunculus, and trichinæ. The microscopic power necessary to define it should be high enough to disclose a red globule, but not higher. An account of the life cycle of the *filaria sanguinis hominis* has been given by Manson, who regards the mosquito with dark and light markings on the body and legs as the intermediary host. The mosquito feeding at night imbibes filaria with the blood of its victim, and then retiring to some shady place near water digests the meal, and matures its ova. The insect then betakes itself to water, and deposits boat-shaped masses of eggs. While in the stomach of the mosquito the filaria undergoes a metamorphosis, becoming possessed of an alimentary canal, rudimentary organs of generation, and crown of *papillæ* which serve as a boring apparatus, and

[1] *Report of San. Com. with Gov. of India.*

enable the filaria to leave the body of the mosquito, when the insect dies after depositing her eggs. The favoured few filaria thus surviving are drunk with water by human beings. From the human stomach it bores its way into the thoracic duct or some lymphatic, and is followed by one of the opposite sex, obedient to sexual instinct. The progeny passes into the circulation to await the chance of a friendly mosquito. The supposed parent worm of the *filaria sanguinis hominis* appears to have been first discovered by Bancroft, in Australia in 1876, and is said to attain the length of three- and a half inches. The worm was also found in 1877 in the urine, by Wucherer of Bahia, and by Lewis in 1877 in the scrotum of a young Bengalee affected with nævoid elephantiasis. Manson states, 'It is now known that the parent worm lives in the lymphatics.' The embryo while *in utero* stretches its oval chorional envelope to form the sheath in which it lies extended as seen in the blood, and when the stretching is complete the filaria is easily borne into the lymph-stream. But when from any cause, such as acting as a foreign body and exciting inflammation, the parent worm becomes unhealthy, the oval embryo are prematurely expelled. These being seven or eight times the diameter of the filaria in its elongated sheath, act as emboli to the nearest lymphatic glands, whence ensues stasis of lymph, regurgitation of lymph, and partial compensation by anastamoses of lymphatic vessels. This brings about hypertrophy of tissue, and may go on to lymphorrhœa, or chyluria, or elephantiasis, according to the site of the obstructed lymphatics. The death of the parent parasite in the afferent lymphatic may give rise to an abscess, and the frequency with which abscess of the scrotum or thigh is met with in Chinese practice is, in Manson's opinion, attributable to this.

Manson also offers an explanation why many observers have failed in finding the filaria in elephantiasis and lymph-scrotum. He says the habits of the filaria are nocturnal, to

suit those of the mosquito, and he regards this as an instance of the wonderful adaptive power of nature. During the day the filaria rests in the minute branches of the pulmonary artery, and periodicity of presence (and periodicity of fever) is accounted for. Manson does not incline to the view that there is a diurnal reproduction of embryos with a corresponding destruction.

The objections to Manson's theory are many and grave. He admits that filaria are found in the blood of many persons not suffering from any apparent disease; in fact, he states that in China 10 per cent. have filaria in the blood, as also dogs, beasts, and birds. Manson expressly limits the noxiousness of filaria to occasions when immature birth of the ova takes place, fully developed young worms passing readily into the circulation. It is objected that too much is assumed in supposing the parent worm liable to miscarry, although Manson considers he had sufficient evidence in two cases that such abortions had happened. Another is the reputed absence of boring apparatus, excepting the crown of papillæ mentioned by Manson (p. 243), which, while perhaps sufficient to allow of the filaria extricating itself from the dead body of a mosquito, it is difficult to conceive how with only such an apparatus the worms make their way from the human stomach into the thoracic duct or lymphatic vessels. Personally I do not believe in the origin of elephantiasis in any of the causes named, excepting one, viz., inflammation of the veins, to which I also add inflammation of the lymphatics, generating, as a consequence, deteriorated blood, or an anæmic condition which shows itself as elephantoid disease rather than by its more ordinary manifestations. In this view I am supported by the fact of the malady most usually presenting in those localities where anæmia is most ripe; in the poor and badly nourished rather than in those well-to-do; among the rice-eating population rather than among those who live on better diet; in agricultural

labourers and others who are exposed to great vicissitudes of temperature. I am also encouraged in this belief by the resemblance which many cases of elephantiasis of the leg bear to *phlegmasia dolens*. Why some persons become the subjects of elephantiasis from blood dyscrasia, and others suffer in a different manner cannot be explained, and must be referred—as we are obliged to refer many other variations of disease—to differences of constitution and temperament, which are not understood.

Treatment.— In the earlier stages, especially if there is fever, the disease may be somewhat controlled by anti-periodics, as quinine and arsenic, and by tonics, as iron and its preparations. Iodides should also be administered. Eliminatives, purgatives, and diuretics are occasionally of use. A milk diet has been recommended; and in all cases, to obviate any scorbutic deterioration, a due proportion of anti-scorbutic vegetables should form a portion of the diet. Leeches, bandaging, frictions with iodine ointment, biniodide of mercury ointment, or what is still better, iodised oil may also be used, and a small issue has been proposed. Scabs and crusts may be removed by poultices. But the best remedy is, as Fayrer states, removal to another climate—the European to Europe, the native to the upper provinces. As a preventive measure Manson says, 'If people in countries where filaria is endemic would cover their water-jars with netting sufficiently fine to keep out mosquitos they would never get filaria in the blood, or the disease it produces—elephantiasis.' The importance of pure drinking water is not questioned, but whether this simple measure would prevent elephantiasis is more than doubtful; for even admitting filaria to be the cause, the germs may be in the water before it is placed in the jars. Therefore Manson should have added boiling and filtering to his recommendation, which, however, the native will not take the trouble to do. Manson also says, persons known to harbour the parasite ought to

sleep under properly constructed mosquito curtains, and so lessen the chance of mosquitos conveying the filaria from them.

When the part affected has become a useless encumberance, and the general health has not materially suffered, the question of removal by the knife may be entertained. Peet asserted that removal is the only means of curing elephantiasis, and most operators agree that after removal the fever ceases. Statistics, however, are much required to show the after-career of those on whom operations for elephantiasis have been performed. I have some reason to believe that their life is short, and that if the disease does return they succumb to some obscure internal malady, or to pernicious anæmia. Successful, or at least temporarily successful operations for scrotal elephantiasis were first performed by Delpech, Liston and Larry in Europe, by Clot Bey in Egypt, and by Esdaile, Allan Webb, Shircore, Brett, Ballingall, in India. But the excessive hæmorrhage which took place rendered it a hazardous proceeding. If evidences of exhaustion are not so strongly marked as to forbid reasonable hopes of recovery, the operation is usually undertaken. The enlargement is raised and compressed so as to force every drop of blood into the circulation, and the best method of accomplishing this is by the use of the elastic bandage fixed by a jean band round the waist, and to two loops passing round the thighs, as proposed by Partridge[1] of Calcutta. Then the groins should be examined for herniæ, the tumour raised by assistants or a pulley, and Fayrer directs an incision over the course of the cord and the dorsum of the penis, which should be dissected out. The testicles, which are often atrophied or calcareous, should be sought for and gently disentangled, after which the tumour may be cut away with a long catlin, no attempts being made to save flaps of integument in which the disease may return. All arteries must

[1] *Ind. Med. Gaz.*, Jan. 1875.

be immediately ligatured, and brandy cordials and laudanum should be at hand to guard against syncope, and diminish shock to the system. Sometimes a clamp has been used for the double purpose of keeping up existing herniæ and of preventing hæmorrhage; but this has appeared to favour an after gangrenous condition of the cut tissues. That the penis and testicles may sometimes be effectually preserved there is evidence in the report of cases where these organs cicatrised over, and procreative power was regained; but if the tumour is very large, and the penis and testicles lost in its substance, the attempt to save them will not often succeed, while the hæmorrhage may not be controllable. Sometimes one testicle may be preserved and the other cannot. Where insurmountable difficulties exist, we have authority to make a clean sweep, and remove the whole.[1]

Ligature of the femoral artery has been advocated for the cure of elephantiasis of the extremity, but Fayrer reports unfavourably; the swelling being reduced so long as the patient remains in bed, but returning afterwards. On the other hand, Bryant [2] has reported relief and cure following ligature. But the remark of Fayrer previously quoted may be recollected, that the elephantiasis of Europe is not that of the East.

[1] Webb, *Field Notes on Amputation.*
[2] Medical Society, *Lancet*, March 10, 1883.

CHAPTER XVI.

FEVER.

Sir J. Paget in his Bradshaw Lectures observes, there are certain diseases which have attracted much notice within the present century, and which have been regarded as new diseases, but are probably only deviations from some well-recognised typical disease, the older form becoming rare, the variations more frequent. This is in a great measure true with regard to fevers which have been divided and subdivided into so-called distinct forms, when in reality they are only deviations from the typical disease. Thus systematic writers describe a variety of fevers marked by distinctive characteristics. But the majority of fever attacks fail in affording all the features required for undoubted differentiation. Although a *typical* case of either so-called typhoid, typhus, or remittent is readily recognised by certain special signs and symptoms, it is yet, as Alexander Smith [1] truly remarks, 'often unfortunately at the beginning impossible to determine how the case may end.' A fever commencing as a common cold or ague may terminate as remittent, typhoid, typhus, or even as cholera (*vide* p. 91). A fever commencing as remittent may end as typhoid; a fever commencing as typhus may end as typhoid. There may be all the symptoms of typhoid excepting the fever being of a remittent type; with all the symptoms of typhoid or typhus

[1] *Cholera and Fever from a New Point of View.*

recovery may take place by crisis;[1] and, as Lyons[2] has shown, all fevers may present the relapsing type. Many medical officers[3] have recently noted in the 'Indian Medical Gazette' and elsewhere, mixed or undefined phases of fever. Thus Dr. Waters,[4] Professor of Pathology, Bombay, remarks that, 'A certain proportion of the remittent fever cases presented some characteristics which, though in several respects not those of either remittent or typhoid, yet partook of both.' Similar instances are noticed on the authority of various other medical officers, by Deputy Surgeon-General Hewlett.[5] It is the same also in semi-tropical countries.

Dr. Chaplin[6] describes a malady, prevalent at Jerusalem, under the name of 'malarial typhoid,' in which the symptoms partake of several varieties of fever, but are wholly referable to no one type. In America it is the same as referred to at p. 253. In Europe, also, Dr. Pedlow[7] notices continued fever of essentially the same nature as enteric and remittent. Hertz also speaks of 'continued malarial fever.'

Maclean observes there is reason to believe that when fever has followed the consumption of unwholesome water at sea, it has sometimes been *not* malarial, but enteric. As regards epidemics, the difficulties of diagnosis are exemplified in the description of the fever which devastated the Hooghly, Jessore, and Pernah districts of Lower Bengal, reported on by Elliot, Palmer, and Anderson in 1863; by Cutliff's report on Meerut fever in 1868; by Taylor's report on the same districts in 1869, and by many other later records of epidemics of fever which might be referred to.

In an article on the 'Diagnosis of Indian Fevers,' published some years back,[8] I wrote, 'It is not too much to

[1] Harris, *Ind. Med. Gazette*, April, 1885. [2] *On Relapsing Fevers.*
[3] Ruxton, Doig, Lucas, Farquhar, Sullivan, Harris, &c.
[4] *Report on Enteric Fever in the Bombay Presidency*, 1883.
[5] *Ibid.* [6] *Lancet*, Sept. 19, 1885. [7] *Medical Times*, July, 1884.
[8] *Indian Annals of Med. Sci.*, 1879.

assert that in very numerous instances no one can positively designate the exact nature of the disease. The conclusions arrived at by one adept would be different from the convictions of another equally qualified to judge. . . The presenting signs and symptoms are not sufficient to prove an infallible guide. Even when the history of the case is forthcoming, the disease cannot always be placed in accordance with the nomenclature of the day. And still more unlikely is a correct solution when, as so frequently happens, fever-stricken patients of whom no history is obtainable are brought to the physician. Thus we find cases regarded as typhoid when they are not true enteric, and cases regarded as remittent when they are something more.' Sir J. Fayrer, who describes various typical fevers, states, 'A considerable amount of climatic fever occurs in the tropics, when the symptoms so closely resemble those of typhoid that they may be, and often are, mistaken.' It is the tendency of fevers, especially in the tropics, to shade off into one another as it were, which has led to the perplexing variety of names under which remittent and typhoid have been described, to the number of some sixty and eighty respectively, and hence to the difficulty experienced by most observers in reconciling their observations with the dicta of the great exponents of fever types. As Da Costa[1] states, 'Certain it is that many divisions are uncalled for. Nature, herself, by the readiness with which she permits even essential traits to be interchanged or to become blended in the same attack, proves that even groups are not widely distinct. The classification of fevers is to a great extent matter of speculation.' The truth of the latter remark is exemplified by the instructions given to the medical officers of the Suakim Expedition that 'All diseases with elevated temperature should be regarded provisionally as enteric fever, which in actual practice does not invariably conform to the temperature range

[1] *Medical Diagnosis*, 1884.

which has been far too arbitrarily laid down by medical authors.'

The classification of fevers being thus to a great extent matter of speculation, hence the idea from time to time broached that all fevers excepting the eruptive forms are variations from one form, the eruptive forms or exanthematic fevers being not true fevers, but diseases accompanied by and causing fever. In support of this it may be advanced, *first*, that the influence which leads to fever may manifest itself in every shade of expression, cholera being regarded by some authorities as the ultimate result of the highest intensity of that malarial poison which under other circumstances excites the mildest form of ague. *Secondly*, that all febrile disturbances from a common cold, affecting the Schneiderian mucous membrane, to severe typhoid affecting the intestinal mucous membrane, are characterised by the same phenomena, viz., more or less chill, succeeded by more or less pyrexia; or in other words, the first apparent event in the chain of sequences constituting any kind of fever is functional injury of the nervous system, followed by functional inactivity of the most important organs of the body, proceeding in some instances to organic disease. *Thirdly*, if we are to define a continued fever as one in which the temperature is the same throughout the twenty-four hours, there would be no such thing, for continued fever shows diurnal variations, although no remissions. *Fourthly*, continued fever often does not differ more from the remittent type than the latter from the intermittent. *Fifthly*, it is found in practice that the one type frequently changes into the other, a reversion to the original type being common. Sullivan[1] states, 'During a long residence in tropical climates I have never yet failed, by making the proper inquiries, to trace back a case of remittent or continued fever to its original intermittent type;' and Haspel proceeds even further when he states that fevers,

[1] *Diseases of Tropical Climates.*

dysentery, and diseases of the liver constitute an indivisible whole, under the dominion of a single cause. *Sixthly*, we might divide and subdivide other diseases in the same way as fevers have been manipulated; for the latter do not differ in their phases more than the former, as for example cholera, scarlet fever, small-pox, of all of which there are many varieties. *Seventhly*, Christison (and others), who did not regard all fevers as one and the same disease, was nevertheless led by his experience to regard fevers as not necessarily marked out from one another by well-defined boundaries, but as shading off gradually into one another. Even Murchison,[1] who (on paper) so well distinguished one fever from another, stated, 'Pythogenic fever may be said to form the connecting link between the continued and remittent.' *Lastly*, if one bacillus, as asserted by Buchner, although denied by Klein, can be converted into another—the hay bacillus into the anthrax, for example—and if, as many would have us believe, fevers are caused by bacilli, there is no reason why one fever should not be changed into another. From such arguments the conclusions have been drawn that there is no such thing as a specific fever, each variety being a phase of the general state fever, modified into different types by the influence of attendant circumstances.

It has been sought to explain the 'hybrid' or undefined forms of fever, so frequently met with in the tropics, by regarding them as the results of several agents developed from various sources, the fevers varying as the quality and quantity of the deleterious agencies vary, and as the conditions of the systems which are called upon to withstand the onslaught vary. Or, in other words, by regarding them as typhoid, modified by malarious influences, or as malarious, modified by typhoid influences. To this class of cases the terms 'typho-malarial,' 'malario-typhoid,' 'typho-remittent,' &c., have been applied, especially in America, by Flint, Wood-

[1] *On the Classification of Fevers.*

ward, and others. 'Not only,' says the former author, 'may remittent present typhoid phenomena, but the diseases not unfrequently occur in combination, being caused by the combined action of malaria and the special poison of typhoid; the symptoms of the one or of the other variety of fever predominating in accordance with the potency of the exciting cause.' Woodward[1] defines typho-malarial fevers as that class which are caused by the specific poisons which produce both fevers. Other practitioners, in so-called malarious localities, have been accustomed to say that remittent becomes *converted* into typhoid. Others advance that the effect of the aguish tendency in individuals suffering from enteric fever is to lessen the tendency to infarction of Peyer's glands, and so to modify or mask the symptoms of enteric fever. I am sceptical as regards either combination of causes, or metamorphoses, or antagonism. I incline to the view that fevers in their several forms are further developments of the mildest form, and are due to the same causes; just as I regard a catarrh, a tonsillitis, a bronchial affection, and a pneumonia, as further developments of cold or chill, acting on the mucous membrane of the parts implicated.

I anticipate certain objections. It will be stated that malarious fevers are caused by malaria, and to this I reply that the existence of malaria as a special poison has not been proved (*vide* p. 259). It may be argued that the bacillus formed in typhoid fever is the germ causing the disease. But inoculation has not proved such to be the case.

It may be advanced that the spirillum found in the relapsing fever originates that malady. But similar spirilli have been found in the blood of those not suffering from relapsing fever. Then there is the prevailing opinion, as supported by systematic authors, that, as quinine benefits malarious fevers, and does no good in other fevers, the

[1] Pepper's *System of Medicine*, 1885.

diseases must be different. Many authors lay stress on the diagnostic value of the benefit which is or is not obtained from quinine. For instance, an author of repute[1] wrote, 'When the attack is obscure at the commencement, has resisted quinine in full doses, and has a duration of from twenty-five to thirty days, very little doubt need remain of the disease being typhoid fever, notwithstanding that the eruption is absent, and the abdominal complication little marked.' A more glaring exemplification of the often insurmountable difficulties besetting diagnosis could scarcely be adduced than the obligation to rely on the effects of a remedy which only enables us to come to a conclusion at the termination instead of at the commencement of the malady. The very difficulties of diagnosis which are sometimes unsurmounted even after death, seem to point to one genus rather than to distinctive genera. And so indeed does the action of quinine. For a very little experience in the tropics will convince that quinine is not the panacea, even in so-called malarious fevers, which it has been asserted to be[2] (*vide* p. 321). Further, some authorities, especially continental, regard quinine as the best remedy for typhoid and non-malarious fevers generally.

It is curious that many who regard fevers as essentially different in origin and nature, do not hesitate to affirm the transformation of a non-contagious or a malarious fever into a contagious malady. The older authors, as Pringle and Lind, fully believed this, and so do many recent observers. During the last twenty years the different Indian official gazettes show numerous reports on epidemics, in which a malarious origin has been ascribed to an admitted contagious fever.

Carpenter endeavoured to explain this by the results of overcrowding. He says, 'There is something wanting to

[1] Peet, *Principles and Practice of Medicine*, p. 503.

[2] *Vide* author 'On the Value of Quinine,' *Ind. Med. Gaz.*, 1870.

complete the maturation of the germs' (the presence of which it must be recollected is assumed, not proved), 'and to effect their elimination from the body in the infective form. This sometimes appears to be supplied by overcrowding. . . . It is an established fact that under such circumstances malarious fevers may change their type and become personally communicable. . . . Overcrowding means deficient air-supply, and deficient air-supply means deficient oxygenation of the blood, producing an accumulation in the circulating fluids of those waste-products which are normally eliminated as fast as they are poured into it, thus affording a pabulum on which malarious saprophytes become fully developed, instead of more feebly vegetative.'

That the one type of fever theory is not generally accepted is probably in some measure explainable by the fact that fever cannot be judged of, as many have pronounced judgment from the experience of one locality, of one epidemic, of one country, or even of one zone; for fever manifests very different symptoms at different times, places, seasons, and among different sections of a population, and in different individuals of such sections. Why fever presents such various phases as to have led to the division into distinct diseases, is no more explainable than the reason why, after exposure to cold, one person suffers from catarrh, another from pneumonia, and a third from rheumatism. Much, most probably, depends on the temperament and constitution of the individual.

As Hutchinson[1] remarks, 'Peculiarities (which may be hereditary) may affect individuals so far as to modify or minify the course of an exanthematic fever, so as to render its recognition quite impossible;' and if this is the case with fevers ordinarily presenting a characteristic eruption, it must be the case with non-eruptive fevers. It is curious that those denying the influence of constitution on the

[1] *The Pedigree of Disease*, 1883.

phase of fever, do not hesitate to attribute differences of symptoms in various individuals to constitutional differences.

When it is recollected how much liability to disease depending on constitutional differences varies, not only in individuals but in families, and even in races; when it is recollected of how many elements our surroundings are composed, and how intimately they are associated, it may be asked if we are not wrong in attributing fevers to any specific poisons. It appears more probable they are the result of the combination of all the influences to which we are subjected, and that the greater or lesser tendency which we show to take on diseased conditions is the result of all the combination of external influences to which we in our persons, and in the persons of our ancestors, have been subjected.

Light may be radiated, reflected, and refracted in many different measures according to the substance it impinges upon. Similarly, heat may be radiated, reflected, refracted, and absorbed. Electricity may abound or the reverse, and it may be positive or negative. Chemical action means a fresh arrangement of particles of matter, and is constantly going on everywhere. Magnetism is a force which in its behaviour with matter resembles electricity in many particulars. Numerous substances are magnetic, others are dia-magnetic. In the absence of any certain knowledge how we are influenced by electricity and magnetism, it is known that our surroundings in any living locality depend largely on the amount of heat and light received from the sun, and on the presence of more or less moisture. Then there are occult forces in nature of which we know little or nothing. The secondary elements in our surroundings are the various forms of organised life which cover the earth and pervade the air, both vegetable and animal. A sum total of favourable conditions of all these influences, un-

doubtedly tends to health; a preponderance of unfavourable conditions may be the cause of those maladies of which we vainly endeavour to find the material poison.

No one denies that the febrile condition may apparently arise from either exposure to cold or to the sun, or from dyspeptic derangements; all causes interfering with elimination and nutrition. Excessive exertion, whether bodily or mental, has always been ranked as a potent predisposing cause of disease, and especially of fevers, and this is traced to the rapid waste of tissue, whereby the blood-current becomes unduly charged with the products of disintegration. In hot climates the activity of the respiratory process being reduced by the high external temperature (*vide* p. 20), the products of waste tend still further to accumulate in the system. Thus it is at least possible, if not probable, that fevers may be maintained by the formation within the body of material which may be regarded as of the nature of a ptomaine. We resist the work of auto-infection ordinarily in two distinct ways, viz., by the elimination of toxic substances, and by their destruction by oxygen. In all forms of fever through defect of the emunctories this elimination and destruction is perverted or interfered with to the greatest extent in the more severe forms; the exact nature or phase of the disease varying according to the toxic material accumulated in the system. It seems reasonable to place fevers in the same category as traumatic fever after contusion without wound, as gout, as arthritis, as rheumatism, as, in short, diseases the causes of which are formed within us—more readily in some constitutions and conditions of constitution than in others, and more readily under vitiated conditions of atmosphere. And it does not seem reasonable to regard fevers as caused by specific external poisons or germs, none of which have yet been demonstrated. The eruptive fevers I regard as diseases causing fever. The maladies hereafter described I regard as phases of the

disease—fever—which may or may not cause a petechial appearance on the skin.

Malaria.—Among germs I include that mysterious poison called malaria: an agent which, long reputed to be the cause of paroxysmal fever, has been credited with the excitation of a vast number of other maladies, as anæmia, beri-beri, cholera, dysentery, elephantiasis, insolation, paralysis, convulsions, neuralgia, gout, even hysteria, &c. Meredith, Cutcliff, and some others have asserted that it causes sterility in women and impotency in man, and many have endorsed the assertion of Maculloch,[1] that 'Malaria produces a far wider mass of human misery than any other cause of disease.'

But malaria is invisible, imponderable, and not recognisable by any chemical or other test. All that we are taught regarding the characteristics and *habitat* of malaria has been deduced by inference from presumed consequences; these consequences being, as Maclean[2] asserts, 'its pathological action.' It may be, perhaps, contrary to sound reason and judgment to declare that what nobody can find does not exist, for there are other poisons which have not yet been isolated which do exist. But it is in accordance with sound reason to require that the arguments in favour of a specific poison should not be contradictory, that the characteristics and alleged production of the poison should be referable to facts unopposed to each other.

The ancient authors, while evidently aware of the unwholesomeness of marshes, did not advance any theory of a specific poison. That must be credited to Lancisi, who in 1745 attributed malaria to decaying vegetation. This theory soon received wide credence, the hobby being ridden to such an extent that some writers, as Rankin[3] for instance, did not hesitate to express the conviction that every tree was a

[1] *On Malaria.* [2] Quain's *Dictionary of Medicine.*
[3] *Public Health in India.*

'malaria trap,' and that 'every blade of grass in its decomposition may reascend in vapour bearing the poison of its putrefied solution into the atmosphere.' Even so recently as March 1877 Dr. Sullivan wrote (*Medical Times*): 'Malaria is confined to certain localities in low, moist situations subject to constant decay and putrefaction acted on by a hot sun.' But it did not take long even after Lancisi's day to convince the majority that fevers occur not only in the neighbourhood of marshes, but also on surfaces where there is no vegetation, such as bare sands and rocks. As exemplifying how hard it is to kill the theories adopted and taught in the schools, I venture to mention Maclean's explanation of fever on sandy soils. He says, 'In all cases the soil will be found to contain a considerable proportion of organic matter, and water will be found not far from the surface.' Also Dr. Chevers' explanation, who states, 'All decomposable matters which the floods deposit on the surface of the sand, not finding any neutralising agent there, undergo disintegration, and pollute the atmosphere.' But Fridel is still more conservative to the teachings of his youth when he attempts to explain the occurrence of fever on granite sites by the decay of a fungus, which he states permeates disintegrating granite rocks. Others endeavour to account for the occurrence of fever on sand by an underlying stratum of impervious clay. The Walcheren expedition is usually referred to as an example of the kind. Similar explanation has also been offered of fever epidemics in parts of Assam, Bengal, &c. When fevers have occurred on the sandy beds of rivers, the deleterious effects of an underground stream have been theorised. My experience, however, does not permit the acceptance of such explanations even from men as eminent as those named above. The inhabitants of the semi-desert tracts of Western India suffer much from fever, especially in the cold season. The Bickaneer district may be taken as typical. It consists of tracts of sand or stretches

of low sandhills on a substratum of soft sandstone, far below which (as I had an opportunity of ascertaining) is gravel. Water is three, four, or more hundred feet from the surface. The rainfall rarely exceeds seven to eight inches annually, and often does not amount to five. The products of the sandy soil are stunted acaciæ, and rain crops of 'bajree' (irrigated fields being unknown), various kinds of small grass, and a shrub known as 'phog,' used as firewood. This is certainly not the description of country to which explanations as above apply. Again, it is in my experience that fever prevails on the granite sites of Mount Aboo, where there is no doubt that granite rock disintegrates, and that a vegetable fungus grows in the crevices. The granite rock here splits off in thin flakes, under which the vegetable growth insinuates itself. But the quantity of vegetable growth thus decaying would not, probably, on ten square yards of granite, average as many grains, and to suppose that this could contaminate the breezy heights of the mountain is as absurd as Dolomieu's designation of paroxysmal fever, 'la maladie du granit.' As regards fever occurring when troops have been camped over an underlying stratum of clay, or in populations living over unhealthy geological conditions, there will always be other circumstances, as exposure, want, &c., which must be taken into consideration. The great losses among the troops at Walcheren and Carthagena are always adduced as illustrating the influence of unhealthy localities; but a reference to the history of those operations shows that there were numerous other causes of disease besides malaria. Moreover, epidemics of fever occur among troops and among civil populations when there are no such geological conditions.

If malaria were produced in abundance in all marshy districts, there are localities where people flourish which would not be habitable. Nothing can contrast more strongly than the low-lying swampy humid coast districts of the Concan,

and the dry arid sandy plains of Marwar.[1] If it be true that malaria is generated in the greatest abundance in marshes because they contain a high percentage of organic matter, the districts first mentioned should be infinitely more malarious than the latter, which is not the case. A rice-field during some part of the process of cultivation and irrigation may be regarded as the type of a swamp. The drying surface, the decaying vegetation, the minute animal life, and the rich alluvial soil are all present. It is, therefore, rather perplexing to find that those authoritatively enunciating the *habitat* of malaria are not agreed as to whether rice-swamps are unhealthy. In Italy rice-cultivation is not permitted within a certain distance of towns, and numerous official Indian reports attribute unhealthiness to rice-cultivation.[2] On the other hand, Indian officials where rice cultivation is extensively conducted deny unhealthiness from this cause. People live and flourish in the Concan, as may be seen within half a day's journey of Bombay, surrounded by rice-fields, while dispensary statistics show the percentage of aguish maladies is not greater in the rice-producing districts than in other places. The Bombay irrigation authorities appear to be imbued with the idea that neither rice nor sugar-cane, nor any other kind of cultivation, is unhealthy, even when attended with over-irrigation; for the station of Poona, the seat of government during several months of the year, is intersected with cultivation of various kinds at the will of the owners of the land; perhaps on the erroneous principle that the energies of the soil are thus directed to food-producing ends instead of to malaria. In spite of this principle, excessive irrigation, leading to a waterlogged soil, and consequent dampness of the ground, increases disease everywhere. A similar conflict of opinion is found as regards salt-water

[1] *Vide* the Author's 'Marwar, the Land of Death,' *Ind. An. Med. Sci.*, 1378.

[2] *Report of the Royal San. Com. on the Army in India.*

marshes, and mangrove swamps.[1] Again, some authorities have attributed immunity from fever to saltness or alkalinity of the soil, while others refer malaria to alkalinity of the soil promoting putrescence.[2] It is also confusing, and not compatible with the malaria theory, when we find, as Dr. Moir mentioned in his report on Meerut, fever was there referred to the scanty rainfall, and in subsequent years to the very opposite cause—a heavy rainfall. It is also confusing, and not compatible with the malaria theory, when we find one medical officer stating that fevers prevail to the greatest extent at stations on the sea-coast,[3] while another medical writer remarks, 'The neighbourhood of the sea is so far exceptional that such situations are nearly always free from diseases produced by malaria.'[4]

Again another contradiction. Malaria is supposed to lose its noxious properties by passing over water even of small extent. When such evidence is analysed it will be found to resolve itself into the fact that individuals who have been exposed to exertion under a tropical sun, or to the damps of night on shore, have suffered more than those remaining in rest and comfort on board ship. It is curious that those who endorse the statement of malaria losing its virulence by passing over water account for the sickly climate of Sierra Leone by the marshy character of the *opposite* Bulam shore. Lancisi, the father of this theory, contradicted himself, for he states that out of a party who had sailed to the mouth of the Tigris twenty-nine were attacked with ague on the wind shifting and blowing to them over a marsh, so that in this case the intervening water was no protection. It has been explained that the ozone found in most abundance in the air on the surface of water destroys the malaria. But

[1] The Author's 'Malaria *versus* Recognisable Causes of Disease,' *Ind. Med. Gaz.*, 1876.

[2] *Ibid.*

[3] *Army Med. Report*, 1860, p. 391.

[4] *Review Mad. Med. Jour.*, vol. x.

it has been found that ozone exists in the air over marshes, and malaria emanates therefrom.

It is said that trees and groves destroy malaria or prevent its spread. This idea has arisen not from actual facts following on the planting of trees, but principally from assertions which have been made of places becoming unhealthy *after* the felling of woods and forests. It cannot be denied that woods and forests are often a protection, but they protect from other evils. They shield from winds, and they mitigate to some extent violent atmospheric vicissitudes. It must also be recollected that the planting of trees implies attention to the ground in the way of cleanliness, drainage, and the consequent diminution of damp. It is in this manner that the planting of trees may be expected to be beneficial, and not, as theorised, by intercepting or destroying mephitic emanations. Stations in India where trees have been planted with the view of staying malaria (as Berhamper, Peshawur, Gorrukpur, Jacobabad) are not especially noted for salubrity. Recently many trees have been felled at the latter station, under the idea that they rendered the place unhealthy.

Malaria is supposed to be most powerful at night, because paroxysmal fevers very frequently occur after night exposure. But so do numerous other diseases. In many parts of the world, as in India, the fall of temperature after sunset is very marked, and in India after the heat of the day the skin is exceptionally liable to impressions from lowered temperature. Damp is also more decided at night, sudden currents of air are colder, and persons in India generally sleep with doors and windows open, or in the open air—if they shut up they are exposed to heat and defective ventilation—sleepers kick off their clothes during the warmer part of the night, and become chilled as the colder period sets in; they are, moreover, often chilled by punkahs, especially when the puller goes to sleep, and waking commences to pull suddenly over his perspiring victim. Lastly, the vital powers are lowest

when the system is in repose. These are quite sufficient reasons why disease originates from night exposure, without reference to malaria.

It is stated malaria is of greater specific gravity than atmospheric air, and therefore more powerful near the surface of the ground. This has been regarded as proved by the facts of low-lying localities being less salubrious than elevated sites, and of upper storeys (except attics) being more conducive to health than basement apartments or cellars. But low-lying localities are damper from drainage and percolation from above, and from more frequent mists and fogs, while upper apartments and higher localities are more exposed to prevailing breezes and are therefore better ventilated. Moreover, in tropical climates elevation conduces to sound sleep from better ventilation, comparative coolness, and comparative freedom from mosquitos. The man who sleeps well habitually, is better able to resist disease than he who passes disturbed and restless nights, and rises languid and unrefreshed. These are quite sufficient reasons why elevated sites and upper sleeping apartments in the tropics should be conducive to health without the theory that they raise the occupant above the range of malaria. Indeed, we are somewhat contradictorily told by some authorities that malaria ascends by day and falls by night. Also, according to Carviere, it will ascend 400 to 500 feet in Italy; 2,000 in the West Indies; 3,000 in America; 1,000 in California; 2,000 to 3,000 in India. Maculloch indeed asserted, and his words have been frequently quoted, that labourers in some parts of Italy are only safe so long as they retain the erect posture. But the most reckless riding of the hobby is that of Martin, who said that in China when *drunkards* fell down and lay on the ground in a state of intoxication, death was almost inevitable, 'as if the poison from its ponderosity did not rise to a great height, and was imbibed more rapidly from the recumbent position.' A drunken man with a brain

congested by alcohol, is very likely to die when exposed either to the rays of a tropical sun or to the chills of a tropical night, and this without the aggravation of malarial poisoning. It also requires some credulity to accept a statement of Mr. Joseph Hume, recently quoted by Mr. Edwin Chadwick[1] that he found safety to his men from malaria when bivouacking, by having them raised eighteen inches above the surface, which certainly might protect them from damp. When we are told[2] that in Greece and Italy people preserve their health by sleeping on platforms raised on poles; that natives in some parts of India find safety in trees; that the American Indians escape by sleeping in trees; that workmen on the Panama railway sleep in high shanties, &c., we are told indirectly that they escape the effects which would otherwise arise from sleeping on damp earth, and that they thereby escape from damp, animals, and insects, and so obtain refreshing sleep. It is true authors mention ague killing in three hours or by collapse. But fatal syncope occurs from other causes, as pure debility; and rapidly fatal cases as above are very different to the sudden dissolution of healthy labourers or drunken men. When death occurs from collapse during fever, post-mortem examination will generally reveal some old thoracic or cardiac imperfection, or the patient will have been worn out by recurring disease.

It is stated malaria is lessened by cultivation and population, and that the first cultivation or excavation of land is always an unhealthy process. There are however reasons why this should be so, irrespective of the release of malaria. The primary cultivation of land must necessarily be undertaken under difficulty and discomfort. Without efficient shelter and adequate food, and with great physical exertion, sickness would prevail whether the ground was disturbed or not. As

[1] *Ventilation with Air from Superior Layers*, 1886.

[2] Crudeli, 'On Malarious Countries,' *Lancet*, Nov., 1884.

countries become cultivated, damp is lessened, and the inhabitants are proportionately better fed and housed. Sir R. Christison attributed the cessation of ague in Scotland not to diminution of malaria, but to the people being better fed. When Englishmen maintained 'stew-ponds' to supply a fish diet, when rushes were used instead of carpets, when in the absence of glass the night air penetrated freely into habitations, when agriculture was rude and drainage unknown, our forefathers suffered severely from ague, fever, and dysentery. We do not require a specific poison as malaria to explain why diseases should be then more rife. Again, when large excavations for fortifications, railways, or canals have been conducted, and the work-people have suffered, the explanation is to be found in exposure, in working in water, in bad food, or in the class of people employed (perhaps prisoners), rather than in the liberation of hypothetical poison.

Malaria, it is said, may exist in water, and be conveyed into the system by such medium. Many instances are recorded of malarious fevers following and apparently caused by drinking impure water, especially in the Terai. But the very fact of drinking such water implies, either on shooting or travelling in such districts, extraordinary exposure, exertion, and neglect of ordinary sanitary precautions. If impure water would excite malarial fever, the inhabitants of many Indian villages with which I am acquainted would not live through a single season. But the fact is, the inhabitants of such villages do not suffer more from fever than people of other villages where the water-supply is better. There are numerous poisons and diseases which may be introduced into the system by water, and there is no necessity to add malaria to the list, in order that the importance of pure water may be demonstrated.

Malaria, it is said, may be wafted by currents of air, either alone or enveloped in mists, from its place of origin to a distant locality. But if malaria is thus capable of travelling

horizontally, it cannot be of a gaseous nature, for the law of the diffusion of gases would come into operation. This therefore effectually disposes of the theories of those who have regarded malaria as carbonic acid or carbonic oxide (Bonnyman), or carburetted hydrogen (Pickford), or sulphuretted hydrogen (Kirke), or ammoniacal gas, or a vapour (Parkes), or carbonic oxy-sulphide (Schwalbe). If malaria, as is theorised, may be conveyed by winds, it must be an entity, and not magnetic influence (Martin), or electrical agency (Heyne, Munro, Eisenmann), or volcanic agency (Parkin). If malaria is conveyed by winds, it must be something definite and ponderable. Hence the endeavours which have been made by Mitchell, Salisbury, Niemeyer, Crudeli, Klebs, &c., to show that malaria consists of low vegetable organisms. The latter gentlemen profess to have discovered the bacillus malariæ (a schizomycete) in the shape of small rods in soil and in water, and in the sweat and blood of persons affected with fever. The bacillus may be artificially cultivated, and when introduced under the skins of dogs and rabbits, produced something akin to typical fever. Cuboni and Marchiafava found spherical mobile micro-organisms in the white blood-corpuscles, which, they theorised, might be the spores of the bacilli. But the fact that so-called malarious disease has prevailed on every kind of surface appears to have been overlooked or ignored by those writers who have endeavoured to discover the cause in one geological formation, or in the production of one form of low vegetable organism. It is scarcely reasonable to presume that alluvial soils, ferruginous earths, decaying granite, limestone rock, marshes, dry sand, all produce the same kind of poisonous emanation, or of vegetable organism. If malarious poison is really evolved from the earth, it seems reasonable to suppose various geological structures would produce different results, and that therefore the characteristics of malarious disease would differ. Yet malarious disease

prevails on all the structures named, and when typical it presents very similar characteristics everywhere.

But, say the votaries of malaria, there must be a specific poison, for malarious diseases are marked by a special phenomenon—periodicity. It may, however, be advanced that periodicity is a law pervading nature. Day and night, the seasons, the tides, the budding of plants, the falling of leaves are all periodical. So also, in accordance with this universal law, are all fevers. Hectic, for instance, is a periodical fever, but no one supposes it is caused by malaria. Relapsing fever comes and goes with the suddenness and periodicity of a typical ague, but it is held that relapsing fever is in the first instance at least caused by want. There is indeed periodicity in enteric, in typhus, and even in so-called continued fever; in fact, in any fever which the ingenuity of nosologists has added to the cumbrous nomenclature. Intermittent fever may be regarded as the type or likeness of all fevers—in fact of all diseases—to which mankind is liable, from a common cold with its chilliness, heats, and perspirations, to typical typhoid with its characteristic exacerbations and remissions. When writers have asserted that disease, and especially disease in tropical climates, is 'impressed with periodicity,' they would appear to have recognised but a portion of the truth, periodicity being an ever-present and essential condition of all disease, but more marked in tropical climates, where atmospheric periodicity, or change, or vicissitude, is most powerfully felt by the human frame, in consequence of the debility of the skin caused by heat. But instead of being thus recognised as a universal law, periodicity has been vaguely supposed to distinctively mark the exclusive offspring of marsh malaria. Why one malady, as ague for instance, shows periodicity more prominently than others, is still a mystery as great as where the flower hides its scent, or the flight of a bird. But many questions in nature have perplexed theologians, philosophers, and meta-

physicians in every age, and will continue to perplex them to the end. If, however, philosophers had been content to answer problems by reference to a hypothetical agency, such as malaria, our knowledge of many things would have been infinitely less than it now is.

I think we may explain paroxysmal febrile diseases without summoning mystic malaria from its hypothetical home. If we take almost any report of malarious epidemics and closely examine the circumstances, it will be found that the persons so suffering were exposed to other and recognisable causes of disease. Among troops it will be found they were undergoing extraordinary exposure or exertion in a tropical or semi-tropical country, or that their food and sanitary surroundings were indifferent, or that they were exposed to great heat by day and to comparative cold and damp at night—perhaps to over-ventilation of their barracks—or to all such causes combined. Among civil populations similar causes will be found in operation. As regards individual cases of fever, it is seldom that exposure to a chill may not be traced, with probably prior mental or physical depression, and possibly dyspeptic derangements. The prevalence of so-called malarious fevers is most noticeable after the Indian monsoon. To the equable temperature preserved by the cloud-blanket that for four months during the rains has checked radiation, succeed clear skies which at sundown allow the heat of the ground to pass rapidly into space, causing a diurnal range of thirty degrees or more of temperature. But this even does not represent the range to which an Indian peasant, or persons employed in the sun are subjected. During the day such persons probably work under 140 degrees of solar heat, the setting sun leaving them suddenly in an atmosphere of 50 or 60 degrees of temperature. Thus at one hour copious evaporation from the skin is required to maintain the temperature of the body below that of the heated air, while the next hour opposite physiological pro-

cesses are necessitated, and the system must be compensated by a supply of heat from its own inadequate carbonaceous store; and how inadequate that must be will be easily inferred, when it is recollected that two scanty meals of rice, a few tolahs of vegetables, and a little mustard oil, form the daily sustenance of most ryots; and when it is remembered that the heat itself often entirely prevents the appetite and interferes with the digestion of the European, it can scarcely be matter of surprise if these sudden reverses of important functions are repeated in a spasmodic form, *i.e.* in paroxysms of burning fever and shivering cold. It will of course be advanced that those countries where changes of temperature are most extreme, such as Scandinavia and the British Isles, are comparatively free from paroxysmal diseases, while on the other hand regions noted for more equable temperature are the home of intense forms of such maladies. But those thus arguing do not seem to appreciate that excitement and debility of the cutaneous system, and that extreme liability to impressions from lowered temperature which characterise those residing in hot climates. A fall of a few degrees of temperature in the tropics will produce a greater impression on the human system than a fall of many more degrees in the temperate zones, the inhabitants of which are better protected by their clothing and houses than in the tropics. Again, the Indian sea-coasts are usually spoken of as equable in temperature. This is a mistake. The variation between the calm stagnant air of morning, and to healthy persons the refreshing sea-breeze at midday and evening is great, and I attribute much sickness near the coasts to this diurnal change of temperature.[1]

One of the most cogent arguments in favour of malaria is based on the fact of countries formerly malarious being now less so; but the result arises from the population being in better condition as regards food, clothing, and other cir-

[1] *Vide* Author's *Health Resorts for Tropical Invalids*, p. 70.

cumstances (p. 267). Another cogent argument in favour of malaria is based on observation of bodies of men removed from one locality to another becoming the subjects of malarious fever. Such so-called malarious localities will be found to be low and damp, or the accommodation or food or general hygienic arrangements will be found defective.

The fact is, the majority of writers confound climatic and other influences with malaria. Now, if malaria is a distinct and special poison it must be distinct and separate from climatic causes, which imply heat, cold, damp, vicissitudes of temperature, presence or absence of ozone, electricity, and neglected hygiene, from which a separate poison should be completely distinguished. This, however, is not done except in those books which, while describing a typical telluric poison, also describe typical disease. That we do not always meet with the latter, is a patent fact. That we cannot recognise the former except by inference, is admitted by the supporters of the malaria theory. I agree with Dr. Oldham [1] in attributing the origin of ordinary febrile conditions to chill and its consequences, but I regard the more severe types of fever as resulting from something added or formed within of the nature of a ptomaine (*vide* p. 256), being still the remote consequence of the alterations in the system excited by chill, and often aided by peculiarities of constitution and vitiated atmosphere.

In thus casting off the thraldom in which we have been so long held by malaria, I find myself in goodly company. Inman,[2] rejecting the malaria theory, pertinently observed, 'Because a theory is a time-honoured one is no reason why it should not be subjected to the rigid scrutiny of science, and, if found unworthy of credit, it should be exploded.' Burdel [3] regarded marsh-poison as a myth. Knapp [4] does

[1] *What is Malaria?*

[2] 'On Malarial Fevers,' *Brit. Med. Jour.*, 1876.

[3] *L'Union Medical*, 1859. [4] *Researches on Primary Pathology.*

not admit the existence of supposed malaria. Surg.-General Munro [1] states 'there is no such thing as malaria.' Dr. Peacock [2] refers the prevalence of malarious fevers to high temperature and hot weather, and not to specific poison. Dr. Rennie [3] wrote, 'Let mud and malaria alone, they will give no one the ague; it is fresh air, not foul, that gives the ague.' Sir W. Smart [4] attributed the fevers of Hong Kong, not to insalubrity of climate from malaria, but to disregard of sanitary precautions. Dobell,[5] 'We speak of it in a glib style as malaria, but in reality know nothing about it.' Armand entirely rejects the idea of a specific marsh-poison.[6] Aitken [7] says, 'It still remains to be shown that malaria has a substantial existence.' Ziemssen,[8] after considering the usual views of malaria, states, 'The necessary conclusion from all this is that the telluric influences referred to above are not sufficient to account for the origin of malaria.' Dickson [9] regarded malaria 'as a fable.' Bascombe [10] refers disease to atmospheric disturbances, hygrometric influence, &c. Mons. Farge, in his description of the Landes of Gascony, asserts the non-existence of malaria.

Among authors who have written with more especial reference to India there is Dr. Oldham, who, in his able and elaborate work 'What is Malaria?' entirely rejects the theory of specific poison, and refers fever to chill. Dr. Lyons [11] writes, 'The established opinion that intermittent is due to the existence of miasm is not applicable.' Dr. Bellew [12] believes malarious fevers are produced by chill.

[1] *Army Med. Rep.*, 1872. [2] *Medical Times*, 1859.
[3] *The British Commissioners in China.*
[4] 'Diseases of Hong Kong,' *Lancet*, 1861. [5] *Year Book for* 1872.
[6] *L'Algérie Medicale.* [7] *Science and Practice of Medicine*, vol. i. p. 504.
[8] *Cyclopædia of the Practice of Medicine*, vol. ii. p. 584.
[9] *Fallacies of the Faculty.* [10] *History of Epidemic Pestilences.*
[11] *Report on the Native Army of Bengal*, 1873.
[12] 'Fever in the Punjaub,' *Ind. Med. Gaz.*, 1878.

Dr. Planck [1] is unable to resist the conclusion that damp is the principal cause. Surgeon-General Gordon, C.B.,[2] 'If my critic will be so good as to define malaria and malarial disease as distinct from climate and endemic influences, he will confer an important benefit on future investigators.' Surgeon-Major O'Connell,[3] 'Ague is a disease due solely to seasonal or climatic influences.' It appears to me the question asked by Dr. Macnamara,[4] viz., 'Is that which we call malaria the sum of the operations of the various conditions of climate and place by which we are surrounded?' may be answered in the affirmative.

Forms of fever.—I was of opinion in 1874,[5] and I am now of opinion, that there is a mixed form of fever in India which, whether we regard it as a *hybrid* or a *specific* form, requires an authoritative place in the nomenclature in order to avoid the actual impossibility of diagnosis of many and increasing instances, under either one or the other of existing heads, which now leads to confusion, and which has led to the erratic use of many terms, as endemic, enteric, typho-malarial, typho-remittent, &c., and other still less significant names, such as Bombay and Bengal fever and rock fever. I consider the term used by Sir J. Fayrer—'climatic fever'—as the one most applicable, for it does not entail adherence to any preconceived notion, as is the case with such terms as typho-malarial, malario-typhoid, &c., while it is applicable to any locality, which such names as 'tropical' or 'Bengal fever' are not. But I would prefix the word 'undefined' until science enables us to replace such definition by some more precise term.

The least severe form of fever is, I believe, an ordinary cold, the more severe phases being ague, continued fever, remittent, relapsing, typhoid, typhus, cerebro-spinal fever,

[1] *N. W. Provinces San. Rep.*, 1874. [2] *Medical Times*, Oct. 1880.
[3] 'Ague, or Intermittent Fever,' *Ind. Med. Gaz.*, 1884.
[4] *Himalayan India.* [5] 'Diagnosis of Indian Fever,' *Ind. Annals.*

and undefined climatic fever, all of which are observed in India, the last-mentioned being the most common.

UNDEFINED CLIMATIC FEVER.—A considerable proportion, if not the great majority, of the fevers of India seem a mixture of several phases based on a few prominent symptoms common to all, and this whether the fever occurs epidemically or sporadically.

A case of *undefined* climatic fever commences, like all other fevers, with an indefinite period of languor, lassitude, and chilliness, or sometimes rigors, with more or less headache, succeeded by a gradual but sometimes a sudden rise of temperature. From the period when the temperature rises to 101° or 102° till the date of death or recovery is from a fortnight to three weeks ordinarily, occasionally shorter, but often much longer, extending even to 120 days. During this time there are always morning and evening variations, the range of temperature being sometimes as much as 9°, frequent but variable remissions and often amelioration about every ninth day, when the appetite may return and the inexperienced be led to believe there is little the matter. Occasionally also there is complete abrupt cessation. Thus, in the thermometric range the fever somewhat resembles remittent, in the duration of the febrile stage it most resembles typhoid, while in the tendency to periodical amelioration and the occasional abrupt cessation it simulates relapsing. Maculæ may or may not be observed. They may be slight and dubious, and when marked they may present the appearance of the rose spots of enteric or the darker spots of typhus. Diarrhœa, light or coloured, may or may not occur, while abdominal tenderness, tympanitis, and iliac gurgling are equally uncertain. The stools are sometimes light, at others dark. Blood is occasionally passed *per anum*. The spleen is generally enlarged, often also the liver, and not unfrequently there is sallowness of countenance or even jaundice. As in enteric, about the middle of the second week there is

usually delirium, the cerebral symptoms being more akin to those characteristic of enteric than of typhus. But sometimes intelligence remains perfect, and there is no delirium unless the case becomes complicated with pneumonia, which often occurs to natives, especially in the north of India. During the whole attack debility is strongly marked. After death the spleen will be found more or less affected; the liver may be more or less engorged, and there may or may not be ulcers in the lower part of the ileum or of Peyer's glands, but often higher up the passage. But as with the symptoms during life, so with the signs after death, while any characteristic of any phase of fever may be present, any may be absent. The disease may sometimes most resemble remittent, at others typhoid, typhus, or relapsing, yet not be found fairly distinguishable as either, according to the dicta enunciated by the exponents of fever types, but yet the blurred image of all. Any complication or sequelæ which occur during or after any other phase of fever may present during or after undefined climatic fever, but pneumonia is the most common complication.

CONTINUED FEVER.—Simple continued fever sets in with lassitude, chilliness, or shivering, succeeded after a variable period of hours or even days by headache, hot skin, quick pulse, thirst, and pain in the limbs. The bowels are generally confined and the urine high-coloured. The fever is soon at its height, and it then gradually declines, with copious deposits of lithates in the urine, or as commonly is suddenly relieved by profuse perspiration, or perhaps by a critical discharge from the bowels. Generally simple fever runs through all its stages in a few days, when it is spoken of as 'ephemeral,' but it may be protracted for a week or upwards. Sometimes there is throughout great irritability of the stomach with prolongation of the fever for weeks even, and to such cases the term 'gastric fever' has often been applied.

The causes are mental or physical fatigue, errors in diet, followed by exposure to cold, to damp, or to the sun, a combination of causes being most potent. When mental or physical fatigue or anxiety are among the exciting causes, the attack is characterised by greater prostration, simulating the more severe phase of fever known as typhoid. When errors of diet or exposure to the sun are the prominent exciting causes, the ailment assumes a more inflammatory type. This phase has been termed 'ardent' or 'sun-fever,' and chiefly prevails (sometimes with epidemic intensity) in the months of April and May, and in seasons when the temperature is unusually elevated. By many authorities it is regarded as a form of sunstroke, and, as I consider it is caused by heat alone, it is described under 'insolation.'

Both simple continued fever and an ordinary cold, if prolonged, require to be distinguished from the exanthemata and from so-called specific fevers. They are to be distinguished from chicken-pox by the absence of eruption; from measles by the absence of coryza as regards continued fever, and by the absence of rash as regards catarrh; from scarlatina by the absence of sore-throat in continued fever, and by the absence of rash in a prolonged cold; from small-pox by the absence of pain in the loins and eruption; from typhoid by the sudden rise of temperature. From the commencement of so-called remittent continued fever is not easily distinguished, but twenty-four hours' observation will usually mark the difference by the characteristic remission. The difference, however, so far as the two latter are concerned, is more in degree than in reality, for the one may pass into the other.

INTERMITTENT.—In cold climates most authorities agree that the tertian type of intermittent fever is the most common. Annesley and Martin also state the same form was most prevalent in India. But more recent observers, as Morehead, Day, Macpherson, Waring, Ewart, Fayrer, men-

tion the quotidian type as most generally met with, both among Europeans and natives, and this accords with my own experience. There is, however, in India not only every degree of intensity of intermittent fever, but also every variety. Of the numerous modifications those forms known as the *tertiana duplex* and *tertian spuria* appear to be the most common, the former differing from the quotidian in that its paroxysms do not answer to each other singly but alternately, the first fit coming on in the forenoon, the second in the afternoon, the third in the forenoon, the fourth in the afternoon; the latter having longer paroxysms than ordinarily, and consequently inclining to the remittent form. It very frequently, however, happens that intermittent, whether unchecked or interfered with by remedies, assumes such irregular and erratic types as to defy reduction to either of the recognised modifications. The cold stage may be absent or very transient, and at times other manifestations may take its place. Thus one individual is warned of the approach of fever by pain in the back and limbs; another by frontal headache; a third by irritability of temper; a fourth by drowsiness and general feelings of discomfort; a fifth by burning about the eyes. Similarly, there are peculiar variations of the other stages. The cold stage may occur and not be followed by the usual sequelæ. Or the attack may apparently commence with exudation, the prior changes being scarcely recognisable. This occurs most frequently after several severe attacks, when the person is convalescing. A form of intermittent has been noticed, consisting of a slight cold fit, followed by the hot stage, after which the hands and feet begin to perspire most profusely, the other parts of the body remaining quite dry; or the trunk is wet and the limbs dry. This phase with its local perspiration, and hence probably retained *materies morbi*, is usually accompanied by intense headache, and generally greater suffering than attends an ordinary

paroxysm. In other instances a slight rise of temperature, followed by slight diaphoresis, constitutes the whole attack. The tendency to irregular types of ague is most remarkable among debilitated populations and in localities to which the term malarious is most applicable.

TYPICAL AGUE is divisible into the cold, hot, and relaxed stages. The commencement of the cold stage is characterised by languid circulation, feeble action of the heart, languor and sense of debility, with disinclination for exertion. There is frequently drowsiness, inclination to stretch the limbs, often headache, and, especially if the person has taken too much quinine, *tinnitus aurium*. There may also be a sense of præcordial oppression and sighing, or hurried respiration. Then the sufferer feels cold, which may indeed be recognised by another person, and certainly if the thermometer is used, before the patient himself admits it. As the coldness increases the person shivers, the pulse becomes more feeble, the skin becomes pale and corrugated, the features, and even the whole body, appear contracted; the lips, ears, and nose in severe cases becoming bluish in colour. There is often nausea and vomiting, while the tongue is white and dry; the last condition also affecting the fauces, which gives rise to thirst. Occasionally there are copious alvine evacuations, but often there is no stool till towards the end of the paroxysm. The urine is generally colourless. Often pains are felt from the first in the limbs and back. As regards the intellectual powers, attention and recollection are generally difficult during the cold stage, but there is usually no delirium. It may, however, be noted that occasionally the cold stage comes on with drowsiness and stupor, increasing to the degree of coma. This cold stage may last from a few minutes to several hours, a protracted cold stage adding much to the danger of internal congestions. Among debilitated populations the cold stage is sometimes so intense as to simulate the algide condition of cholera, and to

terminate fatally by collapse. Towards the end of the cold stage the inner parts of the body appear to burn, while the outer parts still freeze. The hot stage is characterised by the gradual return of heat to the surface, which may be detected by the thermometer before it is perceived by the patient. Flushes of heat are first felt about the neck and face, and soon the whole skin becomes dry and burning, the temperature rising to 103°, 104°, or even 106°. The face flushes, and the conjunctivæ become injected. There is usually acute throbbing headache, and the pulse becomes quick, full, and hard, while the patient is very restless and irritable. Splenic murmurs may also perhaps be now heard, probably due to dilatation of the vessels, and being analogous to those heard in the uterine sinuses during pregnancy. This stage may last several hours, and is more strongly marked in the robust than in the anæmic. At length the sweating-stage commences by moisture first felt on the face and neck, but soon extending to the whole surface, which becomes drenched with acid perspiration. The pulse now sinks to the natural standard, a feeling of comfort is experienced, and the patient begins to feel in his usual health, although often remaining weak and 'shaky.' During the interval a slight periodic increase of temperature, or in some cases a permanent increase of temperature, may be detected by the thermometer. There may also be more or less periodic exudation of moisture. Such alternations may be detected many days after the patient considers himself cured.

Both during the cold and hot stage there are often frequent calls to urinate, water being passed in increased quantities during the cold stage, but less copiously during the hot, and least profusely during the sweating period. The urine is limpid during the cold stage, but more high-coloured during the hot, and is often complained of by the patient as 'scalding.' Uric acid is increased considerably during the fit, and after it urates are deposited. Chloride of sodium is also

increased during the hot stages to five times its normal amount (Ringer), and phosphatic acid to one-eighth (Nicholson). The sp. gr. varies from 1018 to 1825. In some cases, as noted long since by Burdel, sugar has been found, but this is not an ordinary condition. Albumen, renal casts, or blood will probably indicate some pre-existing tendency to kidney disease. A consideration of the whole phenomena shows the considerable resemblance which exists between even typical ague and ephemeral, or even continued fever.

REMITTENT.—Our knowledge regarding remittent fever is as curious as unsatisfactory. Many, while regarding it as the cause of great mortality among natives, have doubted its existence as an uncomplicated malady in Europeans. Macarthney [1] regards remittent fever as *ague plus* some complication. I regard it as an exaggerated ague, but experience shows there is not necessarily a complication. The paroxysm of pure intermittent fever is always finished in twenty-four hours. When the remission is considerable, but not complete, and when it is distinctly marked by the return of a cold stage, such fever may be rightly termed remittent. But when it happens that the remission is not considerable, perhaps even without moisture or a cold stage, the disease would be more rightly termed a continued fever. The symptoms of a typical remittent are those of ague without any distinct cold stage. As the disease advances the remissions tend to become more indistinct than at first, and as the fever becomes more of the continued type whatever remission occurs always takes place in the morning. The duration of the disease by such recurring paroxysms is from five to seven, nine or eleven days, but it may endure much longer. The days mentioned have been regarded as critical, when either a favourable termination or the typhoid phase results. Favourable symptoms are more distinct remissions, lowering of temperature and pulse, subsidence of gastric

[1] *Ind. Med. Gaz.*, March, 1885.

irritability if present, and copious perspiration. Unfavourable symptoms are those constituting the typhoid condition—great prostration, brief and scarcely observable remissions, a black dry tongue, sordes on the teeth, loose bloody stools, vomiting of blood (black vomit), often yellowness of the skin. It is such cases to which foreign authors apply the term *pernicious*, and which British authors have described as adynamic.

But remittent does not always, or even usually, present typical symptoms. The malady may commence distinctly as an intermittent, which becoming irregular first, then becomes remittent. Or the attack, beginning as a remittent, may end as an intermittent; or double exacerbations may take place. Fayrer states it is sometimes difficult to say whether it should be called remittent or irregular intermittent. In some cases the disease does not appear for a fortnight, or longer, after exposure to the supposed cause. Under other circumstances the symptoms are immediate, and so sudden and severe as to have been mistaken for insolation. In the weakly native there is usually an adynamic tendency from the first; in the robust European it is generally sthenic. In all long-continued cases the malady assumes the typhoid form, and is not to be distinguished from enteric fever. In fact, enteric symptoms do occur during life, and enteric lesions may be found after death, converting the case into the class of *undefined climatic fever*. Maclean observes of the adynamic form of remittent, '*Post mortem*, in addition to the common lesions of malarial fever, reveals ulceration of Peyer's patches.'

Diagnosis.—Although some observers profess to be able to diagnose remittent from enteric, and although I once thought I could do so,[1] I now confess inability, except in typical cases. The slow and insidious commencement of enteric, the difference in the thermometric curve, the pre-

[1] The Author 'On Remittent and Intermittent,' *Ind. An. Med. Sci.*, vol. xx.

sence of abdominal tumidity and tenderness, the iliac gurgling, the yellow stools, the rose-coloured eruption, are all characteristic of enteric. But there are some cases of enteric which commence more or less suddenly, and the thermometric curve, showing gradual rise in enteric and more sudden in remittent, is not an infallible test but only applies to typical cases. In many cases of enteric the abdominal symptoms have little prominence. Enteric may be even attended by constipation, while remittent may be complicated with dysentery or *diarrhœa alba*. The enteric rash may be absent or masked by *lichen tropicus*, or (on the dark skin) similated by flea-bites, or by syphilitic roseola, or by purpuric spots. Even *post-mortem* examination is not conclusive, for affection of Peyer's glands is not a morbid condition peculiar to enteric alone. Goodeve, so far back as 1858, observed Peyer's lesions after death from simple remittent, and, as previously observed, Maclean admits this may be the case.

ENTERIC FEVER.—I prefer the term enteric to the term typhoid, as being more applicable to this typical phase of fever. For typhoid fever is something more than the typhoid state (characterised by drowsiness, delirium, dry brown tongue, and extreme prostration) which we find prevailing towards the termination of so many acute diseases.

Sexton, of Bombay, in an able article on recent views regarding enteric fever[1] remarks, seeing the prevalence of typhoid fever in India, it is impossible not to be struck with the absence of any mention of typhoid by the older authors. But an attentive consideration of the writings of Dickson, Annesley, Geddes, Allan Webb, Martin, and others, leads to the conclusion that the maladies they described were the same as exist now. Authors on Indian diseases were formerly accustomed to divide fevers according to the season of the year at which they occur; thus there was the ardent

[1] *Ind. Med. Gaz.*, 1882.

fever of the hot weather, the bilious fever of the rains, and the congestive fever of the cold season. The first and last it was usual to call continued fever, and the second remittent, although all three were presumed to be produced by the same causes.

It must be recollected that in their time typhus and typhoid had not been differentiated in Europe. It was, indeed, only in 1846 that the distinctions were laid down in Glasgow. What we call enteric fever was previously regarded as a sequel of typhus or continued fever, or called acute follicular enteritis, nervous fever, &c. The old Indian writers fell into the same error (if error it may be called) as writers on typhus before the differentiation of enteric fever. It seemed sufficient to the former to describe the different phases of Indian fever under different forms of remittent, continued, or congestive, as it seemed sufficient to the latter to describe typhoid or relapsing fever under different phases of continued fever.

It is only during comparatively recent years that typhoid or enteric fever has been regarded as a distinct disease in India. In 1856 Morehead stated, 'Jenner's typhoid was unknown in India;' but Morehead described a phase of remittent tending to become continued, then typhoid, and in the last edition of his 'Clinical Researches,' he admitted the existence of enteric fever. The first to mention typhoid fever in India was Allan Webb[1] at Simla in 1842, but he probably referred to the typhoid condition. So apparently did Kirk, who in 1848 wrote,[2] 'At the termination of the rainy season and beginning of the hot season, congestive typhoid fever is abundant all over the country.' The first to recognise and describe enteric fever as it exists among the natives of India was Dr. Ewart,[3] and soon afterwards Dr. Scriven.[4] And the first to attach importance to it as affecting

[1] *Pathologica Indica.* [2] *Trans. Med. Phy. Soc. Cal.*, vol. xvii.
[3] *Ind. An. Med. Sci.*, 1856. [4] *Ibid.*, 1857.

European health was Dr. Bryden, who from his post as statistical officer with the Government of India, had special opportunities of ascertaining the number of deaths which occur among young soldiers in India from enteric. Since the periods referred to, although there are some who have doubted the existence of true enteric in India, the malady has been recognised by too many experienced officers to admit of its being considered an open question. Enteric fever in India, when a typical case occurs, seems in every respect —in its course, terminations, and lesion—the same as enteric fever in Europe.

Deputy Surgeon-General Pinkerton,[1] several years the able surgeon to the European General Hospital, Bombay, states he is quite sure that what we now call enteric fever has existed over fifty years in Bombay, and has been known as the 'twenty-one days' bad Bombay fever.' The same authority informs me of his opinion that the disease is in every respect the same as in Europe, and that it has no connection with malarial fever, taking ague as the type. Also that enteric fever exists all over Western India, killing an enormous number of natives under the name (in the mortuary returns) of remittent, or simply fever. With Dr. Pinkerton's views as to the indefinite existence of enteric fever in Bombay, as to its being the same phase of disease as seen in Europe, and as to its causing great mortality among the natives, I fully agree; but I believe it to be simply a phase of fever, and not a specific disease of either pythogenic or miasmatic origin.

If we regard enteric fever as a specific disease, due to a specific cause, we must arrive at the conclusion that the specific cause is not the same in India as in England, and therefore that the disease may arise from various causes. The theory that enteric fever originates from putrefying sewage, as taught by Murchison, or that it originates from the

[1] Memo. 'On Enteric Fever,' *Bombay Med. Admin. Rep.*, 1880.

poison of another, as taught by Budd, is not explanatory of the disease as it occurs in India. Whether we adopt the view that the symptoms of enteric fever are dependent on the products of decomposition set up by specific organisms in the discharges, whether we regard them as due to the direct action of organisms or bacilli, there are, as stated by Deputy Surgeon-General Hewlett,[1] no grounds to believe that the enteric fever of soldiers is in all, or even in the majority of cases, due to a specific poison derived from the intestines of a previously infected person. On this point Sir Joseph Fayrer[2] states, the causal relations of this form of fever are not in India limited to those which give rise to it elsewhere. And in another place both Fayrer and Ewart remark that it is quite possible the fæcal theory of causation does not cover the whole ground. If the malady were confined to the Presidency and other large towns where there are abundant sources from which pythogenic poison might emanate, the European views of the cause of typhoid fever might be accepted; but what Deputy Surgeon-General Pinkerton states of Kurrachee may be taken as illustrative: 'We have no sewers here, and the barracks are as clean as possible. The excreta are removed to a hollow about two miles distant, where the manure is taken up by the *mallis* for their gardens, which are still further away. The soldiers get good water brought in eighteen miles by the Kurrachee waterworks. They hardly touch milk, and the barrack surroundings are as clean as can be, yet we are hardly ever without some cases of enteric.' Neither does Pettenkofer's ground water theory help us in the least, for whatever may be the case in Munich, as with cholera, so with fever in India, there is no relation with the height of the ground water and the prevalence of either disease, which prevail where water is eight and where water is 800 feet from the surface. Em-

[1] *Official Report on Enteric Fever*, 1883.
[2] *Climate and Fever of India.*

merich, of Munich, drank from the filthiest ditches with impunity. Natives of India who sleep over the sewers do not suffer. Indian sweepers who remove the nightsoil daily are no worse off than other people. In short, the origin of typhoid fever as held in Europe is not applicable to typhoid in India. Dr. Budd's theory, that it is derived from some pre-existing case of disease; and Dr. Murchison's theory, that it may be generated anew by the decomposition of sewage, do not explain Indian attacks. We cannot trace it, as it appears to have been traced in Europe, to the previously sick; to generation in sewers, either spontaneously or otherwise; to manure from the fields; to contaminated milk, whether *ab initio* direct from the cow affected with fever, or from admixture with poisoned water; to percolation through soil into wells; to ill-trapped water-closets; to dried up material floating in the atmosphere. And the same may be said of Aitkin's suggestion of typhoid being caused by trichinæ. Neither will the mere fact of bacilli of a peculiar kind being found in the organs of those who have died of typhoid convince that these are the actual typhoid germs, or that they are in any way related to the specific virus until it can be demonstrated that the introduction of the organisms into the body will produce the disease.

Sir J. Fayrer, remarking on the tendency of dysentery to pass the ileo-cæcal valve, suggests that it throws light on the so-called typhoid of India, which may be referred to climatic causes rather than to a specific fæcal origin, for if climatic causes can originate disease in the large intestines it is probable they may do so in the small, especially when the system is disordered by fever. It must, however, be recollected that lesion, eruption, and pea-soup stools may be present from other causes. In the article on dysentery (p. 197) it is mentioned that there is reason to believe so-called bilious dysentery has been mistaken for enteric.

Thus, although the maculæ of enteric are described as

peculiar, consisting of a few slightly rose-coloured spots, the size of a pin's head, slightly raised and pointed, well-defined, disappearing temporarily on pressure, and fading away in two or three days, still the enteric maculæ cannot be definitely diagnosed from some of the early spots, resulting from scurvy or purpura, with any greater precision than these can be told from each other, which is usually impracticable. On the dark skin especially, lichen or roseola, or even flea-bites, may be mistaken for the enteric rash. Maclean admits 'There is nothing very peculiar in the spots of typhoid to distinguish them from others.'

Dr. Waters states, 'The rose-coloured rash is frequently absent in cases of undoubted typhoid fever,' and he has often noticed a well-pronounced crop of reddish spots in grave cases of remittent. The enteric spots are so often absent, so often ill-defined, and so often vary in character, that I regard them as simply a petechial eruption, not of a specific or distinctive nature. Similarly, the so-called specific lesions of enteric are also not always distinguishable from similar appearances from other causes. In enteric the mucus membrane of the ileum generally presents the appearance of acute catarrh, but the chief seats of the morbid change are in Peyer's patches, and in the minute and solitary glands, and there is also enlargement of the mesenteric glands. The diseased condition is divisible into the infiltrative, ulcerative, and suppurative stages. The second stage takes place from the end of the first week to the end of the second, when the last stage commences; so that the lesions present very different appearances at various stages of the disorder. The spleen is also enlarged, and there may be granular degeneration of the liver, kidneys, and heart. It is, however, difficult to account for the stress which has been laid on these lesions in the small intestines as characteristic of enteric fever, for Peyerian ulceration, and ulceration of other parts of the intestines, are by no means the product of one cause or in

association with one set or order of symptoms. Very similar appearances result from various diseases, especially from tuberculosis and dysentery. They have also been found in protracted diarrhœa or muco-enteritis. Gordon pointed out that the lesions sometimes found in cholera are indistinguishable. Morehead long since gave cases of remittent in which similar lesions were marked, and so did Goodeve as previously mentioned (p. 283). Indeed it would appear, as observed by Harley, that enteric fever and its attendant phenomena may occasionally become a part of almost any other condition. According to some authors (Wilks, Moxon, &c.), the peculiarities of typhoid ulcer are sufficient to distinguish it from all other ulcers. In dysentery, with malarious fever, it is stated the lesions are, as a rule, limited to the colon and rectum, and in typhoid to the ileum, or even to Peyer's glands. In dysentery, the ulcers are irregular, with ragged or ill-defined edges, and smaller or larger than those of typhoid. In typhoid the ulcers are round or oval, with even edges and about one inch in diameter. But however this may be in Europe, in India it is not possible to make a distinction between these ulcers. Neither situation nor aspect can be accepted as a certain test. Similarly, with the other morbid changes mentioned as occurring in other organs, all such alterations are met with in other fevers. There is not indeed a single symptom during life or after death which can be regarded as pathognomonic of enteric fever in India. The bacilli found in microscopical sections of the intestinal ulcers, in the mesenteric glands, and in the spleen, liver, and kidneys, after death from enteric, cannot yet be regarded as distinctive.

Influence of Age.—The enteric phase of fever chiefly attacks young persons, to whom it is most fatal, 64 per cent. of deaths occurring in young soldiers in India under twenty-five. This is in accordance with experience of the disease in Europe. Murchison says persons under thirty

are more than four times as liable as persons over thirty, the greatest liability commencing from ten to fourteen, increasing from fifteen to nineteen, then diminishing gradually to thirty, and after that age more rapidly. The susceptibility on the part of the young has been ascribed to the physiological activity of the solitary and agminated glands during youth and adolescence. Sir W. Jenner has remarked that the spleen, the lymph glands, and Peyer's patches, all become less and waste down at the same period of life, about fifty, that the diseases in which these parts are involved, especially enteric fever, cease to be common. As with the elderly so with the very young; babies being singularly exempt from enteric fever; a fact of significance with regard to 'milk epidemics,' since milk forms the staple article of diet of children of tender age.

Influence of residence.—Among Europeans, *recent arrivals* in India have been thought to be most subject, and this again has been considered synonymous with arrival into an infected area, and to the dependence of enteric or some local cause to which older residents have become habituated. But, as Indian statistics are principally derived from the military, and as soldiers arriving in India are of the age most susceptible to enteric, it would seem rather a matter of age, and of constitutions depending on age, than of recent arrival.

Influence of season.—There seem to be two periods of intensity, viz., April and May, September and October. The periods of the greatest heat are not the seasons of the greatest prevalence, which seems to indicate some degree, although not the highest degree, of moisture as a necessary factor, otherwise there would be more enteric fever during the height of the monsoon rains, which is not the case.

Second Attacks.—According to Niemeyer one attack of enteric fever removes susceptibility to the disease, except in rare instances, and the statement is generally held to be

correct by those having the greatest experience of the malady. It has also been stated that a previous attack of enteric renders a person less prone to paroxysmal fever. I was acquainted with a medical officer who acted on this belief, going shooting into the most so-called malarious places at the most unhealthy seasons with impunity, feeling secure from a previous attack of enteric.

Mortality.—Few diseases are so fatal in India as the enteric form of fever. The percentage of deaths from different diseases to the total treated, shows enteric fever near the head of the list, the death-ratio being 18 per cent.

Symptoms.—Typical enteric fever may be defined as a continued phase of fever attended with diarrhœa, and characterised by eruption and intestinal lesion, with frequently enlargement of the spleen. But by the admission of all authorities it appears under diverse forms. *First*, there are instances which have been termed 'walking cases of enteric fever,' or *typhoid ambulans*, when the patient, although suffering from the principal characteristics of the malady, does so in so mild a form that he does not require to interrupt his ordinary avocations. *Secondly*, there is 'mild typhoid,' when the person, although confined to the house or bed, is never in a dangerous condition. *Thirdly*, there is 'abortive typhoid,' when the malady terminates suddenly about the seventh to the twelfth day. *Fourthly*, there is 'intermittent typhoid,' when cases of enteric fever present intermittent characteristics from first to last. Among others who have organised the intermittent form of enteric is Harley, who describes three varieties, viz., the simple, the inflammatory, and the paludal enteric. *Fifthly*, there is the remittent form of typhoid. *Sixthly*, enteric fever may set in suddenly with rigors. *Seventhly*, enteric fever with constipation has been described as a separate form. *Eighthly*, bilious enteric fever, when there is bilious vomiting. *Ninthly*, symptoms referable to the stomach may predominate through-

out, masking other symptoms ; hence one of the terms applied to the disease 'gastric fever.' Hence the large number of names under which this phase of fever has been described.

But a typical case of enteric fever is characterised as follows :—The onset is gradual and insidious. There are feelings of malaise, aching of the limbs, dull headache, loss of appetite, and chilliness, while the countenance expresses languor. The sleep is unsound, and exertion wearisome, but for some days the person is able to go about, thinking there is not much the matter. The bowels are usually constipated, but sometimes relaxed from the first, and the person may be supposed to have ordinary diarrhœa. There may also be from the commencement marked symptoms of stomach derangement, as nausea, inability to retain food, and vomiting.

Most frequently, however, this gastric derangement is not sufficiently marked to become the prominent symptom. Feverish symptoms now appear, preceded generally by a rigor, or at least by chilly sensations alternating with flushes. The pulse, at first feeble, becomes quicker and full, but remains compressible and not tense; the skin becomes hot and dry, and at about the end of the first week or sooner the patient takes to his bed, often complaining of aching of the limbs. The appetite is now utterly gone, the tongue coated, and the bowels loose, while the fever if closely watched will be found to be slightly remittent in type. The urine is scanty and high-coloured, there is increasing restlessness at night, the face is often pale with a pink flush on the cheeks, but the eyes are clear and bright. The diarrhœa continues, and the stools passed are thin and of a yellow colour, sometimes resembling pea-soup. They are also said to be devoid of mucus, containing altered blood and albumen with a large proportion of soluble salts. Before this period the abdomen will probably have become tense and resonant. If the hand is passed over the right iliac fossa the patient's face will probably express pain, and a gurgling may be heard or felt

under the fingers. There will also be more or less splenic dulness. Between the seventh and twelfth days, the so-called peculiar eruption of enteric fever appears on the abdomen, chest, and back, consisting of a few slightly raised rose-coloured spots which disappear temporarily on pressure under the fingers, and fade away in two or three days, being in the meantime succeeded by fresh crops. On the darker skin of the native the eruption of enteric fever appears more like flea-bites, as the distinctive points—viz., the absence of the central puncture of the flea-bite, and the less obvious outline, cannot be so well noted as on the fair skin. This eruption is not likely to be confounded with the small watery vesicles of *sudamina* which occur in most fevers. I regard these spots of typhus fever as petechial, and not special or specific. An abundant eruption of a dark colour occurring suddenly will certainly be scorbutic. The malady has now entered the third week, with fever unabated, with acid perspirations, and with signs of the disturbance of the alvine tract and of the nervous system more unmistakable. But in favourable cases, and especially in children, after the appearance of the eruption a diminution of the fever takes place, and more or less deafness, regarded generally as a favourable sign, may occur, the remissions become more distinct, the diarrhœa lessens, the tongue cleans, pains in the limbs cease, the patient sleeps at night, the temperature decreases, and the appetite returns. In more severe cases, about the middle of the second week delirium comes on, at first slight and only noticed at night, afterwards more constant, intense, and noisy. Usually it is not a wild delirium, but rather a confusion of mind producing rambling thoughts. When contrasted with the mental wandering in other acute diseases, the delirium of enteric is more active than that of typhus, less demonstrative and talkative than that of *delirium tremens*, as aimless as but less continued and violent than that of cerebral inflammation. Usually the patient may be

roused to answer a question, but it is worth recollecting that he is very apt to reply to all queries in the affirmative. In some cases the patient is more restless or even violent, with a tendency to get out of bed. As time advances the tongue becomes dry, red, and glazed at the tip and edges, with a dark state of the posterior part, often with cracks in various directions, while *sordes* form on the teeth. The lips also crack and bleed. There is usually frequent shallow persistent cough and bronchial crepitation. Epistaxis may also now occur. As the time advances, or during the third week, change for the better or worse may be looked for. If the latter, the patient continues to lose flesh and strength, he lies prostrate and often unconscious of what is going on around him, and if the case ends fatally he will become quite insensible, his temperature will rise higher, and he will, with trembling hands, pick and 'fumble' at the bed-clothes, while stools are passed unconsciously, and urine is retained until, the bladder becoming paralysed from repletion, it dribbles away. There is also much jerking of the tendons *of the extremities*, which become colder. A large degree of picking and jerking is suggestive of much mischief in the intestines. Dilated pupils, epistaxis, hæmorrhage from the bowels, yellow skin, muttering delirium, are all unfavourable symptoms, and may terminate in coma or convulsions. Death usually takes place about the end of the third or beginning of the fourth week, but in some instances the case may be prolonged beyond the second month.

In typical enteric the temperature is very characteristic. In the first week there is a gradual and steady daily increase from the normal condition to probably 101°5 in the morning, and 104° in the evening, at which it arrives about the seventh day. During the second week the morning and evening temperatures are more identical, probably 104½° in the morning and 105° in the evening, or even above these figures. In the third week the fever begins gradually to be more

remittent, while, however, it reaches almost the same degree at its exacerbations, being at the end of the week probably 100°5 in the morning and 104° in the evening. During the fourth week there is a gradual fall, the morning and evening variations being at first from 4° to 6°, or the fever may become distinctly *intermittent*. A sudden or irregular rise of temperature denotes some local complications, probably in the lungs, which, especially in natives, are very liable to become congested. A marked fall of temperature not unfrequently precedes dangerous bleeding from the bowels. Sudden variations of the pulse will denote similar complications.

As a general rule, the average course of the pulse runs parallel to that of the temperature, varying daily with the latter. It rises to 104° at the end of the first week, and to 108° in the second week. But the absolute height of the frequency of the pulse is less in enteric than in most other febrile diseases. There are some cases in which the pulse does not become rapid for some time after the accession of the malady. As the person becomes weaker, in which the action of the heart sympathises, the pulse becomes more frequent. If the pulse rises to 120° and remains so, the prognosis is bad, as commencement of paralysis of the heart is indicated. But simply lifting the patient in bed may temporarily increase the pulse to 120°. The relaxed, soft, and easily compressible pulse has already been noticed, features which remain till towards the close. The more the heart's action and circulation diminish, the greater the difference between the peripheric and inner portion of the body. The peripheric portions continue to lose heat as usual, and this heat is no longer replaced, as in the normal state, by the influence of a stream of warm blood leading to the cold extremities.

Enteric fever may be *further complicated* and rendered more dangerous from the accompanying *diarrhœa* being very profuse and exhaustive. There may also be profuse

hæmorrhage from the bowels, known by blood in the stools either mixed or in clots, and usually, as previously referred to, marked even before the blood appears externally by fall of temperature. Perforation of the bowels may also occur usually between the twenty-fifth and thirty-second days, an accident attended with symptoms of collapse and always proving fatal. Often in such cases there is no peritonitis, but great distension of the bowels, which are motionless, with great distress of respiration. Simple tympanitis usually occurs late in the disease, as one of the symptoms of great nervous prostration. Collapse, however, may occur from various causes, as it depends on sudden weakness of cardiac action, which may result not only from perforation, but from profuse hæmorrhage, from shock produced by perforation, or even from violent vomiting, when the temporary weakness of the heart's action passes into paralysis and quickly causes death. The erect posture, inducing momentary anæmia of the brain, may also be followed by similar results. There is also the collapse of defervescence. A sudden decrease of fever or sinking of the temperature, either spontaneous or produced by remedies (as the cold bath) may, by withdrawal from the heart of the stimulus of high temperature, cause sudden weakness of the heart and collapse. Inflammation of the bowels, or peritonitis, may also supervene without perforation. Or the liver, or still more frequently the spleen, may become congested. The intense gastric irritation, noted as sometimes characterising the commencement of the attack, may remain prominent throughout. The lungs may also become affected, denoted by quick breathing, short hacking cough, and crepitation. Other complications occasionally noted are otorrhœa, paralysis of one or more limbs, convulsions or spasmodic seizures.

The duration of enteric fever from the commencement of the premonitory symptoms is ordinarily from three to four weeks, but the attack may be prolonged to the end of

the second month, the fever assuming a distinct remittent type. The majority of deaths occur at the end of the third week. The scrofulous or syphilitic taint exerts a deleterious influence, and adds much to the danger. The period of incubation is said, without sufficient evidence, to extend to three weeks.

Relapse occurs in about 15 per cent. of cases, and usually comes on in the second week of convalescence. The temperature quickly reaches the maximum, and the attack is shorter than the original one. Or the relapse may assume a more irregular form, the first symptoms being return of griping, abdominal pains and diarrhœa, which may lead to peritoneal inflammation or perforation. Relapse occurs less frequently in persons above thirty years old than at lower ages. Relapse has been regarded as induced by a new infection, from the same source as the original infection, from the patient remaining in the same place; but as relapses occur to persons who have been moved, the explanation is not tenable. Relapses have been thought more frequent under the anti-pyretic treatment, but this is doubtful.

TYPHUS.—The term typhus, signifying smoke, or vapour, seems to have been applied to the phase of fever especially marked by a peculiar overclouding of the senses. It seems to have been recognised in India years back by Allan Webb, but both Morehead and Peet denied its existence. Since then it has been recognised by various observers, as Walker,[1] Chuckerbutty,[2] Brown.[3] There is also reason to believe that the 'Mahamurree,' or Indian plague, which ravaged Western India about 1815, from Kutch to the Himalayas, was an aggravated typhus. Recently, Surgeon Rice states, 'The fever so rife in many crowded towns cannot be distinguished from genuine typhus.'

The origin of the typhus phase of fever is supposed to be

[1] *Edin. Med. and Surg. Jour.*, May, 1861.
[2] *Ind. An. Med. Sci.*, vol. xviii. [3] *Ibid.*, vol. xiii.

connected with overcrowding. It may be defined as a continued fever, with less tendency to remissions than typhoid. It occurs at all ages, and the commencement is marked by malaise, headache, and chilliness, or rigors. The onset is less gradual than that of typhoid, but this is variable. It may occur sporadically, but usually presents epidemically among poor, crowded populations, or among prisoners, and is then regarded as highly contagious.

The duration is shorter than typhoid, often not being prolonged beyond the second week, but there may be relapses. Gastric symptoms are less common than in typhoid, but may present. The bowels are usually constipated, and the urine scanty and high-coloured. The temperature reaches 104° or 105° at an early period, and is more sustained than in typhoid, the daily variations to the middle of the second week being often not more than 1° or 2°. In the early part of the third week there is rapid subsidence, temperature sometimes falling as much as 4° in a night. The pulse ranges throughout from 110° to 120°, and is often tense, small and weak. Cerebral symptoms come on soon, sometimes almost from the onset, and they often subside about the twelfth day, with deafness. The delirium is of a low, muttering character. The countenance is characteristic, being dusky, with a heavy, stupid expression, and eyes suffused and watery. The skin is said to emit an ammoniacal odour, but I believe this depends upon escaped urine, and that the ordinary odour is acid like other fevers. There is usually no abdominal pain, no abdominal tenseness, and no diarrhœa, or if so, bilious stools. Splenic dulness is usually present. On the fourth or fifth day a ‘ mulberry ’ rash in the form of small, roundish, dusky, or brown red spots, not raised, appears on the wrists, extremities and trunk, not totally disappearing on pressure, and remaining until the twelfth or fourteenth day. This rash, when present, I regard as petechial and not a distinctive specific rash. The tongue is furred, and the fur cracked.

There is marked prostration throughout, and after a few days decided dorsal decubitus. The most common complication is pneumonia, but jaundice is sometimes present. Death not unfrequently occurs at the end of the first week, and often before the conclusion of the second, and is usually by the 'typhoid condition,' seldom from hæmorrhages, and never from perforation. There are no constant morbid appearances. The most common are a dark liquid state of the blood, and enlargement of the spleen, or liver-congestion.

Thus it will be apparent that typical cases of either typhoid or typhus may be readily diagnosed, but unfortunately, in practice in India, typical cases are the exception as often as the rule.

RELAPSING FEVER, from the peculiarities of its course, has been termed short fever, five-day fever, and seven-day fever; from its occurring among persons suffering from scarcity it has been called 'famine fever,' and from the condition of the blood 'spirillum fever' (Carter). Gastro-hepatic, mild yellow, and bilious remittent, are other names derived from symptoms ordinarily prominent. In former days it was regarded as what it really is, viz., a relapsing form of continued fever or typhus, and not as a distinct disease. It is characterised by a remarkable tendency to recur at tolerably regular intervals, the succeeding attacks becoming less violent, and the intervals between them more prolonged. Relapsing fever ordinarily commences with slight chilliness, frontal headache, giddiness, and prostration of strength, but often the premonitory symptoms are very slight, and they may only last a few minutes, although sometimes present for hours. Then the skin becomes more or less suddenly hot and dry, the temperature rising to 103° at once, with increase of headache, with pain in the back and limbs, loss of appetite, and much thirst. On the second or third day sweating may occur, but without relief of symptoms. Contemporaneous with this sweat, or without sweat presenting,

the temperature may fall a little on the evening of the second, or morning of the third day, after which a prolonged rise begins, to 104° or even 108°, and is maintained, with the exception of an evening and morning variation of 1°, till the seventh day, or till the day of crisis. The pulse follows the temperature, but quickens more gradually at first. It almost invariably exceeds 110°, usually reaching 120° or more, on the third day. There is said to be no characteristic eruption, but Carter [1] figures rose-coloured spots, while mottling, or purpuric spots have been observed, but supposed to be accidental. Carter considers sudamina more frequent than in other kinds of fever. Jaundice is often a prominent symptom, sometimes occurring suddenly, sometimes gradually. At first the tongue is moist, with whitish fur, then becoming dry and brown at the back, and creamy in front, with moist, florid edges. The bowels are most frequently constipated, but there may be diarrhœa. Epigastric tenderness, with vomiting of dark material (sometimes urgent) general abdominal tenderness and engorgement of liver (in 60 per cent. of cases) and of spleen (in 70 per cent.) may be expected. The urine is scanty, high-coloured and cloudy, from mucus sometimes containing albumen, and when there is jaundice, bile. Severe shooting pains are felt in back, limbs and head, and delirium may occur at any period, the latter being usually of an asthenic character, and sometimes resembling the effects of dhatura poisoning (Carter). The face sometimes presents a livid or bronzed appearance, with weary, haggard expression, shown even in infants, and regarded by Carter as indicative of the blood being much charged with parasites. Bronchial irritation occurs in about half the number of cases, and some amount of pulmonary congestion in most.

From the fifth to the seventh day, although in rare cases deferred to the ninth or eleventh day (Carter), there is an

[1] *Spirillum Fever*, 1882.

abrupt cessation of all the symptoms, generally attended by critical perspiration, and occasionally with diarrhœa, epistaxis, hæmaturia, or uterine hæmorrhage. The perspiration has sometimes a peculiar, musty odour, and contains rods of bacillus, but no spirilli. The febrile symptoms are now absent for a few days, the tongue becomes clean, the appetite returns, and the person may declare himself well, but there is usually a certain amount of flying pain, languor and exhaustion, although the patient may go about and even gain strength. The temperature is lowest, about 97°, immediately after the crisis, when the pulse is highest. Then there is a gradual change in their relations during the next six or seven days, the pulse becoming slower than natural, and the temperature slightly higher, when there is a sudden return of all the symptoms. The relapse lasts from three to five days, when the fever again abruptly declines. Sometimes a second or third, or even a fourth relapse occurs, but each interval is longer, and each attack shorter.

Several varieties are mentioned by Carter and others. It may commence as an attack of ague; it may assume a remittent character. It may be abortive, or shorter than usual, or presenting no relapse; or there may be latent relapse, or the attacks may terminate gradually by lysis instead of by crisis. There may also be intense jaundice and black vomit, which has been described as congestive, or bilious relapsing fever.

There is no specific lesion. Visceral alterations are of a general congestive and degenerative character found in other fevers, but congestion of the intestinal mucus membrane and vascularity of Peyer's patches is sometimes noticed. Starvation and destitution are the conditions with which this phase of fever is most usually associated. But starvation and destitution do not always excite this phase of fever, for during the famine years, 1868–69, in Rajpootana the type of the generally prevalent fever was remittent and not re-

lapsing.[1] Like all other phases of fever, it is aggravated by overcrowding, want of ventilation, especially as regards emanations from the sick, and all other insanitary causes. When originated, it is considered to be communicable to persons who have not been subjected to want, and is therefore regarded as contagious. The mortality is about 18 per cent. of the attacked, and the period of incubation has been stated at eight days, but without any sufficient foundation. The spirillum Obermeieri is often found in the blood and saliva of those affected with this type of fever. The worms appear in the form of thin threads, showing corkscrew-like movements. They are usually only found in the blood during the height of the fever, disappearing when the fever declines. It has been stated there is no recognisable difference between this spirillum and a worm known as *spirillum plicatile* found in water. Vandyke Carter states the detection of spirillum in the blood is the test in all outbreaks of fever of doubtful character occurring in jails, &c., and he regards the abrupt cessation of the fever and disappearance of the spirillum as distinctive. But there is the fact, as stated by Lewis, that the spirillum has been found in the blood of those not exhibiting any signs of relapsing fever. We therefore cannot attribute relapsing fever to this spirillum.

Diagnosis.—Relapsing fever differs from enteric in the suddenness of its onset, the absence of the characteristic abdominal symptoms and eruption, the absence of the localised iliac tenderness and of the peculiar diarrhœa, in the absence of the well-marked red tip and edges of the tongue of typical enteric, and in the sudden cessation of the symptoms. It differs from typhus in having a higher temperature and quicker pulse at the onset, in the absence of the typical eruption, of the heavy aspect and of the low delirium of typhus, and in oftener presenting the jaundiced condition of the skin. It differs from remittent in the absence of

[1] Author's 'Famine and Fever in Rajpootana,' *Ind Med. Gaz.*, 1870.

remissions. It differs from all as shown by the thermometric charts of typical cases. But on the other hand the relapsing type may pervade any other Indian fever.

CEREBRO-SPINAL FEVER.—*In some cases of enteric, of undefined fever, of relapsing fever, of typhoid and of typhus, symptoms of spinal affection appear.* There may be contraction of the muscles of the neck more or less convulsive, also of the respiratory muscles and sphincters, convulsive cough, and cutaneous hyperæsthesia, extending over some portion of the body or along the spine, where also there may be pain, tenderness, and a sense of weight or pressure. Paralysis more or less confirmed of some limb may also present. Sometimes such symptoms are noticed during the course of the fever, at other times not till convalescence.

These symptoms, combined with the fever, are very similar to what has been described as a specific malady under such terms as *cerebro-spinal fever*, *cerebro-spinal typhus*, *epidemic spinal meningitis*, *typhoid fever of a cerebro-spinal type*, *purpuric fever*, &c., which are described as showing the following symptoms. It may commence very gradually with malaise, chilliness, headache, undefined pains, nausea, or vomiting. Or it may appear suddenly with rigors, intense headache, vertigo, constant vomiting, epigastric pain, pains in the back of the neck, back and joints, accompanied by marked prostration and pyrexia, often also photophobia. However it begins, the development is characterised by stiffness of the muscles of the neck, which spasmodically contract, so that the face is turned to the head of the bed. Sometimes the contractions may extend to the muscles of the spine, but there is seldom (if ever) tetanic arching. These contractions and pains are aggravated by movement. There is also delirium characterised by muttering, incoherent talking, and restlessness, sometimes becoming violent. About the third day cutaneous eruptions appear, in the severe form always of a petechial character. Or roseola or vesicles may present,

the latter frequently as an herpetic eruption on the lips about the sixth day. The temperature throughout runs a varied course, from 102° to 105° or higher. The cyclic oscillatory curve of typhoid is distorted by sudden and irregular variations, as seen in meningitic inflammations. The pulse is frequent but also variable. The tongue is coated white at first, afterwards becoming drier and brown. Hyperæsthesia of some part of the skin is common. The knees are often drawn up to the abdomen, which is retracted. Sometimes the bowels are constipated, sometimes relaxed. The urine at first deposits lithates, afterwards usually contains albumen or blood. The duration of the disease is variable from hours to days. The complications are paralysis of one or more limbs, most frequently the arms; purulent infiltration of the eyes, most frequently the right; acute articular inflammation, which may terminate in purulent effusion; hæmorrhage from the nose, bowels, or kidneys. The termination is usually by the typhoid condition.

There is, however, a mild form of the malady, so slight as to be mistaken for rheumatism of the cervical muscles; or other of the symptoms named above may be more or less feebly marked. Assistant-Surgeon Dadachanji[1] describing an epidemic at the Nara jail, Sind, mentions two forms, the explosive (*meningite foudroyante*) and a milder or subacute form.

Now the symptoms of confirmed cerebro-spinal fever do not differ more from the cerebro-spinal symptoms which are generally admitted as sometimes occurring during enteric and typhus, than two cases of enteric often differ from each other. The points of distinction between cerebro-spinal fever and typhoid are said to be[2] the different form and irregularity of the fever, the more abrupt invasion, constipation, constant vomiting, slower or variable pulse, less heat of

[1] *Bombay Med. and Phy. Soc. Trans.*, 1886.

[2] Drummond, *Diseases of the Brain and Spinal Cord*, 1883.

skin, the early eruption, the different character of the eruption, which is red and not rose, the facial herpes, the hyperæsthesia, and the nervous symptoms. From typhus, in addition, the earlier appearance of the eruption without any mottling of the skin. From both the occasional discovery of exudation of a purulent character in the fissure of Sylvius, on the surface of the pons, and on the posterior surface of the spinal, cervical, and lumbar regions. The points of similarity between the diseases are, however, even more striking. They all may attack the comparatively young, and spinal fever, like typhus, principally occurs in crowded localities. They may all commence suddenly or gradually. All are characterised by marked prostration. All may be accompanied by diarrhœa, or constipation, or vomiting. All may terminate in a few days or longer, all may show an early eruption, and all may be characterised by nervous symptoms. The distinction between a red and rose-coloured eruption is too fine for practical demonstration, while facial herpes is an eruption which usually accompanies a common cold, an ague, and often any kind of fever, and is therefore unreliable as a distinctive sign. Moreover, the eruptions, excepting the herpes, are, I believe, as in other forms of fever, petechial and not specific. As regards the post-mortem appearances said to be found in cerebro-spinal fever, I do not consider purulent infiltration about the brain or spinal cord more suggestive of disease originating in such parts than the occurrence of hepatic abscess or articular abscess during fever, is suggestive of the disease originating in such organs. If cerebro-spinal fever is not the same disease as typhus and typhoid, it is evident both the latter maladies are sometimes marked by the characteristics of the former. I regard cerebro-spinal fever as caused by a morbid determination to the nervous centres, instead of to the follicles of the intestines as in the enteric phase.

The complications which occur most frequently in the

different phases of fever have been noted under the various headings, but there are complications common to all fevers, the most important of which are now mentioned.

1. Sudden Syncope, or collapse, may occur during the progress of any phase of fever, especially when the patient is debilitated, or when in former days he was treated by spoliative agents, as bleeding, leeching, antimony, and purgatives. Syncope most frequently occurs towards the close of the exacerbation, or during the remission, or in ague during a prolonged cold stage. When the feeble pulse, tremulous hands, and wandering mind evidence great debility collapse may be dreaded, and particularly if the patient is suddenly and imprudently raised to the upright posture. In individuals where old chest-disease exists, the danger is also increased. Either affection of the heart, pleuræ or lungs, embarrassing the respiration, will, especially in debilitated subjects, increase the tendency to collapse. The existence of fatty degeneration of the heart materially increases the danger of death from syncope. Death suddenly occurring in the cold stage has been attributed to the right side of the heart becoming paralysed by over-distension by venous blood, but often there will be found some existing acquired constitutional defect.

Hence the necessity of inquiring into the previous history of the patient, and of exploring the chest, in order that double precautions against sudden movements and the assumption of an erect posture may be enjoined where evidence of former thoracic affections exists. It has been remarked that the depression caused by a violent blow on the abdomen more nearly resembles the febrile collapse than any other morbid condition, both probably depending on a disturbance of the functions of the organic nervous system. My experience, however, leads me to believe that fatal collapse is generally connected with old thoracic changes, but the depression resulting from abdominal complications will

doubtless favour the syncopal tendency. However this may be, it is appalling to find a patient whose condition may have been regarded as favourable, suddenly present a thready pulse, sunken features, and cold surface, as indications of a quickly fatal termination.

2. Cerebral Complications.—The least severe appears to be nervous irritation of the brain or membranes, or both. I use the term to express a minor degree of excitement, not appearing to depend, as evidenced by the absence of pathological signs, on either congestion or inflammation. It appears to be caused by deranged and failing circulation, and is demonstrative that the brain participates in the general failure of the vital power. The condition is mostly noticed in weakly individuals, especially in delicate and anæmic European females. It is attended with headache, incoherent rambling, and eventually drowsiness, but the countenance is rather pale than flushed. This species of delirium is generally present during the exacerbation, and may commence with the first paroxysm, but is more frequently delayed to the second or third.

When *congestion* is present the symptoms are more decided and violent, the countenance is flushed, and the conjunctiva often injected. This sometimes occurs during the cold stage, but more frequently accompanies the hot stages of paroxysmal fevers, declining during the remission. As the disease progresses, and in the continued phase of all fevers this congestion is liable to terminate in effusion and coma, which may come on gradually after a longer or shorter period of drowsiness, or less frequently it supervenes suddenly. Drowsiness sometimes appears, especially in the remittent phase, unpreceded by delirium. But drowsiness under such circumstances is not so dangerous a symptom as when it follows delirium, although of unfavourable import, as liable to terminate in coma. In most varieties of head affection, gastric symptoms and vomiting may be

expected, and although gastric symptoms may arise *per se*, when combined with head symptoms they are generally dependent on the cerebral condition. As a general rule head symptoms in sthenic persons will be more severe the sooner in the course of the disease they occur. When they appear in the more advanced stages they are usually attended with adynamic symptoms.

3. The Adynamic, or Typhoid Tendency.—When this occurs it answers to the *febris perniciosa*, or pernicious remittent or intermittent of foreign authors, and it is not improbable that the algide condition, so much dwelt upon, was often attributable to lowering treatment. Bad forms of remittent fever have been described under a multitude of names, as 'scrobutic remittent,' applied when petechial swellings and hæmorrhages were present; *remittens febris comatosa*, when rapid coma, simulating apoplexy, has occurred; *febris algide* comprising the syncopal dysenteric and choleraic varieties; *febris maligna, typhoid, putrid*, &c., when early typhoid symptoms supervene. According to my experience the bad forms of remittent, to which the term *pernicious* is applicable, are in India usually characterised *first* by the fever tending to become rapidly continued, and then typhoid. It has been remarked that in ague a double exacerbation sometimes occurs in twenty-four hours, and in such instances the febrile condition is very liable to become continued. One exacerbation may, however, be so prolonged as to run into the time when the remission should occur, which in such instances is not distinguishable. About the third or fourth day, or sometimes not till the seventh day, the typhoid condition sets in, perhaps accompanied by petechial spots which cannot be distinguished from the eruption of enteric fever. Or *secondly*, the early stage may be characterised by great prostration, uncertain remissions, a quick feeble pulse with marked tendency to hæmorrhages, and this may be expected when there is a

scorbutic taint. Or *thirdly*, pneumonia may appear to be the principal complaint. Or *fourthly*, the cold stage may be so intense and prolonged as to approach the algide condition of cholera. There is reason to believe that some of these cases have been mistaken for cholera.

4. CONVULSIONS generally present towards the termination of febrile attacks, and in many cases happen in the interval between delirium and coma, and towards the termination of an exacerbation. I believe convulsions are more likely to occur in those whose systems have been previously saturated with alcohol. As convulsions are frequently witnessed in the course of remittent, the cause has been attributed by many authors to malarious saturation. But convulsions occur in the course of other fevers, especially cerebro-spinal fevers, which are not regarded as due to malaria, and also to persons living in non-malarious localities. Children are especially liable to convulsions during the progress of intermittent, often occurring in the earlier paroxysms, and sometimes appearing to occupy the place of the first stage. Occasionally *rigid spasms* occur instead of ordinary convulsions.

5. THORACIC COMPLICATIONS are most frequently seen among natives, particularly in the cool season, and in the northern provinces. Congestion of the lungs, often passing on to hepatisation, is in my experience the most common thoracic complication in all phases of fever, and especially in more decidedly paroxysmal phases. The reason is obvious. During the first stage congestion takes place in the delicate lung tissue, which is not entirely removed during the subsequent remission. When the congestion is great, so-called pulmonary apoplexy or pulmonary hæmorrhage may occur, and suddenly prove fatal. Cough, pain, rusty sputa, and the customary symptoms of pneumonia are seldom prominent symptoms, especially in natives. Frequently the patient does not make any complaint referable to the chest, and the

existing condition may escape notice. Hurried respiration is very evident in the later stages, but at first there is little more acceleration of the pulse than the febrile stage would explain. But, on the other hand, in adynamic or pernicious remittent the pneumonia may mask the fever. It is believed that epidemics of fever have been regarded as epidemics of pneumonia. As a rule, the lower posterior portions of the lungs first afford stethoscopic evidence of disease, but sometimes it is first evident in the neighbourhood of the mammary regions. Generally when pneumonia is present the lips and tongue are more than ordinarily parched and dry, especially noticeable in phases of fever, such as remittent for instance, where a moist tongue is usually found, at least in the first stages. The frequent obscurity of the symptoms of pneumonia, especially in the native, renders frequent examination of the chest necessary. It should also be borne in mind that a lung embarrassed by old pleuritic adhesions is more liable to congestive distension than one having free pleural surfaces. Hence the previous history of the case may be such as to lead from the first to suspicion of the probability of pulmonic complication.

Bronchitis occurs more frequently in natives than in Europeans, and under precisely the same circumstances as pneumonia. Its approach is also insidious, and the symptoms are seldom well marked. It generally commences with catarrh, and like pneumonia results in the fever, if of the paroxysmal type, becoming prolonged, and without distinct remissions. But the glassy white expectoration of bronchitis is a more constant symptom, especially in the native, than the rusty sputa of pneumonia. The clear sound on percussion marks the freedom of the lungs, and distinguishes from pneumonia. The sibilant or sonorous rhonchi of bronchitis will also scarcely be confused with the crepitation of pneumonia. But it not unfrequently happens that the former terminates in the latter.

Pleurisy is not so frequent as either of the two former complications. But pleurisy in the native does not appear to be attended with the acute stabbing pain characteristic of the disease in the European. Hence the former more frequently neglect to mention the first indication. Excepting in three instances, I do not recollect hearing the friction sound of the first stage of pleurisy in the native. Pleurisy occurring during fevers arises generally from some accidental cold or chill, or from incautious use of the cold bath, and is not ordinarily excited by febrile exacerbations. Unlike the lung tissue and bronchial mucous membrane, the pleura is not congested and strained during the febrile accession, and thus a potent exciting cause is absent.

6. Abdominal Complications.—The great majority of the complications of fevers in India are abdominal. Martin considered this may be the principal cause of the tendency to collapse so frequently observed. The liver is liable to congestion in every phase of fever, but most liable during the progress of paroxysmal fevers. Or enlargement may take place very gradually, consequent on repeated attacks without any decided symptoms of congestion. As either of these conditions prevail the symptoms will be more or less actual pain, tenderness, or uneasiness in the right hypochondrium, less being present when passive enlargement takes place.

Spleen engorgement is also liable to arise during the progress of any kind of fever, and especially during paroxysmal fevers. In some instances the spleen suddenly enlarges, the organ which could not be felt or seen to be enlarged one day being fully visible the next.

The complication, or sequelæ of diarrhœa or dysentery, is very frequent in all fevers in India. There is first the so-called specific diarrhœa of enteric, and more frequently there is diarrhœa or dysentery caused by the congestion which fever, and especially paroxysmal fever, causes in the abdominal organs generally, and in the portal system and intestinal

mucous membrane particularly. But abdominal affections may consist of mere irritation, as from accumulation in the large intestines. However originating, the irritation may result in dysentery. It has frequently happened that dysentery during fever epidemics has assumed an epidemic intensity, so that it has come to be regarded as the cause rather than the effect, and has been attributed to malaria. As a general rule bowel complaints occur, not on the accession of fevers, but after some continuance or several paroxysms. Those who previously suffered from dysentery, hepatic disease, or spleen, or who are tainted by scurvy, scrofula or syphilis, are more liable to dysentery during fevers than others who have enjoyed robust health.

7. JAUNDICE.—When symptoms indicating affection of the liver exist, *jaundice* not infrequently follows. But in many instances the same condition occurs without prior or accompanying symptoms attracting attention to the hepatic region. Jaundice is most likely to present during the relapsing phase of fever; it is also frequently present during the remittent phase, and is not forbidden by either typhoid or typhus. During both the two former phases of fever I have known extreme icterus come on suddenly during the course of a night, or even in two or three hours. These sudden cases are seldom attended with appreciable symptoms of hepatic affection. When it occurs gradually it is generally attended with tenderness below the ribs. Jaundice, like dysentery, has been known to prevail epidemically during the prevalence of fever, the latter having been sometimes regarded as the effect. When head symptoms occur after the access of jaundice it has been questioned whether they are due to the circulation of the elements of bile, or to the progress of the fever. It has, however, been observed that head symptoms arising from the jaundiced condition occur most frequently in that variety of icterus accompanied by yellow atrophy, not very frequently seen in India. Still the

presence of bile-material in the blood of a fever patient must be regarded as a grave aggravation of the danger. The occurrence of jaundice during fevers has led to the term *bilious remittent.* In this form of the malady jaundice often appears from the outset, and vomiting of dark-coloured matter is an early symptom. The urine is often coffee-coloured, owing not to bile but to blood. In such cases the adynamic condition is always early and markedly prominent. Occasionally epidemics of bilious fever have occurred, when the remarkable absence of pyrexia led to the diagnosis that the cases were merely jaundice.

Bilious remittent has often occurred among prisoners in jail, and has been regarded by some observers as yellow fever. This bilious remittent, or the conditions described under *the adynamic or typhoid tendency* (p. 308), have all respectively been regarded as *jail fever*, the fevers of Indian jails having sometimes shown the symptoms of the one condition and sometimes of the others; often being complicated also with scurvy (p. 313).

8. Hectic.—Both intermittent and remittent phases of fever are occasionally complicated with *hectic*, for phthisis, formerly supposed to be so rare among natives of India, is now admitted to be a very frequent affection, although the disease is somewhat different to that observed in Europe. The extent to which tubercular disease occurs in the coloured races has become evident rather from post-mortem than from direct diagnosis. There appear to be two distinct forms of phthisis in India, the latent and febrile. The former runs its course without any symptoms, except emaciation, without cough, pain of chest, expectoration, or hæmoptysis, at least in the earlier stages. Suddenly febrile symptoms set in, quickly followed or accompanied by evidence of pulmonic affection. In other instances hectic occurs without prior emaciation. Frequently there are short intervals of apparent convalescence succeeded by acute

febrile and inflammatory symptoms. A superficial observer, meeting with one of these instances of hectic, would be liable to consider the case to be so-called malarious fever, but emaciation should always excite suspicion, and lead to stethoscopic examination. Hectic may also be induced by suppuration in various parts of the body, and if such is found to exist the probability of the disease being hectic instead of intermittent will be great. In Indian dispensery practice, people will often apply stating they are suffering from fever occurring every day, and mothers especially will bring their children with a similar complaint, neither mentioning the probable existence of a large boil or abscess on some hidden part of the body. So often was this the case that I habitually inquired if any wound, ulcer, or abscess existed. But there is no doubt that hectic and remittent or intermittent may be present in the same person at one time, showing themselves as two distinct affections. The climatic fever may occur every or every other day, aggravating the hectic on the days of its accession. Or it may demonstrate its presence at a different time on the same day. If in a person suffering from hectic the temperature rises to 104 or more, the supervention of some other fever is most probable, the temperature of hectic rarely rising above 102.

9. Scurvy.—The probability of the existence of the scurvy taint during any fever can never be ignored in India. This union of scurvy and fever has caused a considerable number of the some 230 (or thereabouts) names by which typhus, typhoid, relapsing, and remittent have been described. Whether fevers are distinct diseases, or whether they are variations from one stock, they are one and all rendered doubly severe by the scurvy taint. They more rapidly assume typhoid conditions, and there is a greater tendency to fatal hæmorrhages.

10. We only require the addition of Syphilitic Cachexia to the scorbutic taint and fever, to render the trio a com-

bination more destructive than any other diseased condition. The syphilitic taint alone is only less detrimental than the scorbutic alone, and tends to produce similar results.

11. ALBUMINURIA has been regarded as both the effect of the fever poison and the effect of the fever process. I regard it as the latter, and consequent on that granular degeneration which occurs in the kidneys to a greater or less extent during the progress of all fevers. Its import must be judged of by the amount of albumen present, indicative of the renal mischief which is preceding.

12. THROMBOSIS arises from the languid state of the circulation, and from increased coagulability of the blood, and may occur in any variety of fever. The symptoms of thrombosis are those of arrest in the circulation, and they differ according to the vessel affected. Embolism may also occur in the arteries as a further result. If the blocked artery supplies important organs, symptoms of arrest of function of the part supplied by the artery will follow, as paralysis, dyspnœa, coldness of extremities, or pyæmic abscesses may result, especially of the liver.

13. INFLAMMATION OF ANY GLANDS may occur, and parotitis is not an infrequent complication.

As *sequelæ* there may be prolonged debility, or the varied conditions understood under the terms anæmia, insanity, phthisis, paralysis.

The indications of treatment in any case of fever may be stated as follows:—To reduce excessive cold or heat. To ensure sufficient but not too excessive excretion and elimination. To afford tone and strength to the semi-paralysed nerves and system. To relieve distressing symptoms. To counteract local complications. To, we are told, in case of malarious disease, neutralise the specific poison setting up the fever.

Very little is requisite in the way of treatment for the less severe forms of fever, known as a common cold, or catarrh, or ephemeral fever, or even for simple continued

fever. An aperient, if necessary, and a diaphoretic, as the *liquor ammoniæ acetatis*, may be rightly given; but the great curative is time, in the progress of which the fever will subside. Similarly, with the more severe forms of fever, the disease will come to an end, and it is the physician's business not to attempt to cut short the fever by so-called specifics (of which there are none) but to recognise the *indicatio morbi*, and to use such measures as will best enable the patient to outlive the malady. The means formerly recommended for the cure of fevers possess only historical interest. Venesection, by which it was long believed fever could be cut short, is now never employed. The same is true to a large extent of emetics, although for natives especially I have often found an emetic beneficial at the commencement, by unloading a burthened and foul stomach, by getting rid of a quantity of bile, and by promoting diaphoresis in ague. Again, at the present time we do not expect to cure fever by chlorine water, or the mineral acids, although acidulated drinks are often grateful to the patient.

Calomel, once so highly esteemed as a certain cure for fever when it produced salivation, and even recently recommended by Liebermeister to cut short typhoid, cannot be sanctioned, except as perhaps an occasional purgative at the commencement. A similar remark applies to the mineral salts, such as sulphate of magnesia. Iodide of potassium, either alone or combined with tincture of iodine, formerly much used by continental physicans, has gained no reputation in India. Neither can fever be aborted by quinine, or by quinine in combination with digitalis, and the same may be said regarding the cold water treatment.

At the same time it is admitted on all sides that as the principal recognisable danger from fever arises first from injury during the cold or congestive stage, and secondly from the deleterious influence of high bodily temperature on the tissues, so the abbreviation of the cold stage, and to a

certain extent the abstraction of heat during the hot, are the most important practical measures. The *first* indication (required most in agues) is accomplished by promoting the warmth of the body in every possible manner. The patient should be at once put to bed, covered with blankets, and have hot bricks or hot-water bottles put to the feet. He should drink freely of hot tea, or congee, or cold (but not iced) water if more agreeable. A pan under the bed containing hot ashes is a useful and handy means of promoting warmth. If depression is great, diffusible stimulants are indicated. Emetics are sometimes desirable in the first stage of ague when there is nausea or inclination to vomit, and when the attack has come on shortly after a meal. Mustard and water is the best emetic in such cases, as it tends also directly to warm the patient, as does indeed the action of vomiting. But the practice of administering purgatives or emetics in every case is objectionable, as their operation disturbs the patient, and may subject him to chill at the critical period of the passage of one stage into another.

The *second* indication, the abstraction of heat during the hot stage, is best effected by lowering the circulation by the use of the bath, by the use of wet sheets, by the application of ice, and by digitalis, or by other anti-pyretic remedies, such means being most required in those fevers partaking of the continued type.

The success of the treatment by the cold bath depends, as Dr. West[1] has well observed, on the clinical skill which recognises the proper circumstances for its use. It cannot be disguised that the cold bath under certain conditions is liable to induce or to aggravate internal complications. It has been stated that a person need never die simply from high bodily temperature, so long as the cold bath is available. Pyrexia implies increased tissue-metabolism and increased oxidation. Reduction of heat of body results in

[1] *Lancet*, March, 1884.

diminished metabolism, and as the demand for oxygen is lessened the respiratory action falls, the action of the heart is improved, the liability to cardiac failure is lessened, while the abeyance of the febrile process results in the more natural performance of the secretory and excretory functions, and in the more ready assimilation of nourishment. Admitting all this, I am not prepared to advocate the continued use of the cold bath as an anti-pyretic agent, 'so long as the temperature rises above 103°, severe bronchitis notwithstanding; unless in the presence of some counter-indication, as the occurrence of intestinal hæmorrhage.'[1]

Coupland[1] states he has rarely seen any serious collapse occur as the result of cold baths, but has often considered it necessary to administer a stimulant, and that shivering as the result of the immersion has necessitated a curtailment of the process. I, however, consider any tendency to collapse as adding to the danger, and shivering as an indication that the cooling has been carried too far, and as suggestive of some internal mischief presenting afterwards. Moreover, I consider that the ptomaine causing the several phases of fever (*vide* p. 258), must be eliminated or destroyed before recovery can take place, and the cold bath, by interfering with or stopping these processes, may do injury. The cold bath in my opinion can only be judiciously used to such an extent as to diminish the evil resulting from heat and excessive metabolism, while not preventing the elimination or destruction of the poison causing the fever; a certain amount of metabolism must go on.

Liebermeister recommends, as soon as the fever has declared itself, cold baths at 68° Fahr., or 75° for weakly patients, repeated as often as the temperature rises to 102°; and so do various Indian practitioners, but I should be sorry to pursue this practice. The advocates of the cold bath treatment caution that brandy should always be at

[1] 'On the Cold Bath Treatment of Enteric Fever,' *Lancet*, March, 1884.

hand as a means of preventing syncope, and I fear the risk incurred in the majority of instances more than counterbalances the benefit derived in the minority. Therefore, as previously stated, the systematic use of the cold bath depends on the clinical skill guiding the choice of remedies, and *ex cathedrâ* rules cannot be laid down. The objections to the cold bath are the shock at first inducing internal congestions, followed by reaction inducing syncope, and the check it gives to elimination; and it is also stated relapses are more frequent after its use. Even its advocates regard it as a doubtful remedy if the disease has made much progress. While not prepared to condemn it as 'always bad practice and fraught with the most dangerous consequences,'[1] I believe the caution which should guide its employment is almost prohibitory of its use. It has been stated that, besides the reduction of temperature, the cold bath is useful in another manner, as 'cold kills germs.' But it has yet to be proved that germs are the causes of fevers, and it has to be proved that any temperature compatible with life is destructive of germs. Although the cold bath will reduce the temperature two or three degrees at once, and although the temperature may sink still more afterwards, while the temperature of life remains the germs will live. Instead of the cold bath, I prefer placing the patient in a bath having a temperature of 95° Fahrenheit, which may be gradually cooled down to 85° or 70° by the addition of cold water. If the patient's skin has a temperature of 103° or so, the contrast between that and 85° will be sufficiently decided to reduce the temperature without too great a shock, two, three, or even four degrees.

Mr. Rustomjee Khory[2] states: 'The natives of India, owing to the practice of bathing in hot or warm water, suffer much from cold or shivering if immersed in cold water, and

[1] Cobb, *Lancet*, March, 1884.

[2] *Principles and Practice of Medicine*, 1885.

the dread of its effects prevents them from using a cold bath when recommended in fever.' If the patient is weak, he should be put into a sheet as in a cradle, and lowered by assistants into the tub of water. After immersion for a few minutes the temperature to be gradually lowered. The patient should remain in the bath fifteen or twenty minutes, or not so long if he begins to feel chilly, and then be rapidly dried and put back into bed. It will usually be several hours before the temperature rises as high as it was before using the bath. When this occurs another bath may be used. Although Liebermeister and other advocates of the cold bath treatment do not regard such complications as bronchitis or pneumonia prohibitive, hæmorrhage or decided weakness of the heart's action is so regarded by all. I would not, however, recommend even the modified manner of using the bath above sketched when there are lung complications, or decided congestion of any internal organ. Cold sponging with vinegar and water may then be used to the body and limbs alternately.

Some continental authorities, as Naunyn, restrict the cold water treatment entirely to typhoid fever, drawing a line between increase of heat and the disease causing it, and asserting that in other kinds of fevers cold baths do no good. It is difficult, however, to imagine that remedies calculated to reduce bodily heat can be beneficial in one disease and not in another, increase of heat being the principal phenomenon in both.

Wet sheet packing is applicable to fever cases where the temperature remains between 102° and 103°. The person is wrapped in sheets saturated with cold water, and then covered with blankets, while ice may also be applied to the head. The operation may be safely continued for an hour or more, the sheets being re-saturated when they become warm. After the sheets are removed the patient should be covered with blankets, when he perspires as if in a vapour

bath. This may be used when the cold bath would be dangerous. The application of cold or ice to the head has always been a desideratum during fever. But the application of cold or ice-bags to the supra-clavicular regions is more serviceable. There are many large superficial veins near the neck, and by the direct application of cold to them the temperature of the blood is lowered, and this may be usually done with advantage.

The next most important means of reducing the temperature is by quinine. I do not however regard quinine as possessing all the specific value with which it has been credited. Agues, which quinine is most credited for curing, like many other maladies, have an undoubted tendency to terminate at least temporarily in health; and that they would do so in by far the larger number of instances is a proposition which none can well dispute. But it may be said they do not end so quickly when treated by other agents. This generally received idea I somewhat doubt. I am aware of the existence of tabulated statements apparently showing that persons treated by quinine recover more rapidly than those taking different so called anti-periodics. On the other hand, I have seen patients recover speedily and well without a particle of quinine, both from intermittent and remittent fevers. Bigoted Brahmins and others in Rajpootana, while soliciting the attendance of medical officers, have frequently declined European medicines; yet I have known them recover as thoroughly, and certainly as quickly from both remittent and intermittent as though they had been saturated with quinine. Moreover, I have been obliged to treat patients without this agent, and the cases have terminated as successfully as in other instances, when large doses of the alkaloid had been given. It is very strange that the most powerful, or indeed the only, specific we possess should be for the cure of maladies which are naturally intermitting. When the medicine is taken and the patient recovers, the

medicine is too often credited with the cure which really should be attributed to the *vis medicatrix naturæ*, and thus quinine is daily honoured with additional favour. So it was in former years with mercury, with bleeding, with purgatives. Some years back Hare[1] wrote, 'Salivation is an antidote to malarious fever. The instant a patient's mouth is sore the fever leaves him.' As now with quinine, practitioners in bygone days would scarcely have dared to permit a patient to incur the supposed risk of disease without the treatment then in vogue. It has been advanced by Ewart and others, that the undoubted diminution of European death-rate from fever, is evidence of the powers of quinine. This seems, however, fallacious. The mortality among European soldiers, our chief statistical source, is lessened as regards every disease. This results from better habits of life, from better control of the canteen-issues, and therefore less intemperance, from greater attention to personal hygiene, from better barracks, from the careful general sanitation now practised, from the relinquishment of the mercurial and bleeding practice, but perhaps more than all from increased invaliding and facilities for visiting Europe. But we are told quinine neutralises malaria. Reasons have already been given for doubting the very existence of malaria, and if there is no poison there can be no specific action on it. Probably no one will now assert that all the various remedies from time to time brought forward as anti-periodics have in reality the slightest power over fever. There is scarcely a drug which has not been vaunted as possessing anti-periodic properties, and this has simply resulted from subsidence of the malady after the remedy has been taken. Thus narcotine and charcoal, arsenic and cobwebs, opium and chiretta, fuming nitric acid and 'nim' bark, strychnine and nitre, carbolic acid and berberry, hyposulphites and black pepper, alcohol and coffee, *cum multis aliis*, have all been lauded as

[1] *Ind. An. Med. Sci.*, No. 2, p. 468.

remedies for fever. Paroxysmal fevers have subsided after the exhibition of all, *ergo* all are anti-periodics. Space will not permit a *résumé* of experiments made with the view of deciding on the action of quinine.[1] But I believe the action of quinine to be stimulating in small, sedative in larger, and congestive and poisonous in heroic doses—therefore somewhat similar to the action of opium and alcohol, both once of reputed efficacy as anti-periodics—and by its sedative action diminishing heat. It has also a certain antiseptic influence. But I do not credit quinine with specific properties. The frequent failure of quinine in its supposed specific action has led to the theory, as advanced by Maclean, that some blood depurant is necessary in combination when the alkaloid fails in curing paroxysmal fever. As a general rule, diaphoretics and diuretics are recommended. But it so happens that the skin and urine are the routes by which the *materies morbi* or effete matter is passed from the system, during or after febrile disturbance. The perspiratory process terminates the paroxysm, and with the albuminous fluid many other constituents must escape, while the urine, increased in quantity, contains a greater proportion of uric acid followed probably by a deposit of urates. The remedies used with quinine are those promoting the flow of the two excretions, and they would act in similar manner without combination with quinine. In an article on 'The Value of Quinine,'[2] in which the whole subject is fully considered, I quoted some fifteen writers who have expressed an opinion adverse to the specific action of quinine. I do not assert that quinine is of no utility in fever, paroxysmal or otherwise, but I say it has no specific power, and that its employment should be conducted with such a view of its power. It should therefore be regarded rather as an adjunct to the treatment than as

[1] For a digest see Author's article 'On Fever,' *Ind. An. Med. Sci.*, vol. xxi. [2] *Ind. Med. Gaz.*, 1874.

the principal means of cure, and especially so in remittent or prolonged febrile conditions. I advocate using tolerably large doses of quinine in all fevers during the intermission or remission, or at the time the temperature begins to lower. The doses should be such that more than the primary stimulating effect may be obtained without the results of poisonous doses. Quinine in large doses may originate convulsions, coma, or fatal collapse. Epistaxis, headache, flashes of light before the eyes, præcordial oppression and dyspnœa, are among the less violent effects. Noises or singing in the ears are the first effects of large doses. The latter signs should be accepted as the guide to the quantity to be taken. But in practice there is a very considerable difference respecting the amount of quinine which individuals demand or tolerate. In persons of sanguine or nervous temperament, a given quantity of quinine will generally produce more effect than in persons of opposite constitution. Roundly speaking, it may be said that strong and large men bear more quinine than small and delicate persons, and females do not bear quinine so well as men, absorption being possibly more rapid in the systems of the former. Five, eight, or ten grain doses every three or four hours, I have frequently given, but the clinical skill of the practitioner must be the guide to the choice. Larger doses are, I think, unadvisable, as tending to induce congestive action in the brain. Indeed I believe that when cinchonised patients recover, they do so independently of, and perhaps in spite of, the supposed remedy. By cinchonised patients I mean when the quinine produces roaring in the ears, trembling of the hands, flashes of light, &c., which effects Liebermeister and others require should be produced. I do not recommend quinine during the excess of temperature of any fever, not from the harm it may cause, but because I believe little or none of the medicine is then absorbed. I do not hesitate using quinine when complications are present. Even in head complications I do

not hesitate giving smaller quantities for the sake of the tonic stimulating effect, but I avoid the sedative results, which are in fact synonymous with congestion.

When quinine is administered, idioscyncrasy must not be forgotten. There are persons who cannot take half a grain of quinine without suffering from urticaria, sore-throat, or coryza. The hypodermic method of giving quinine was, I believe, first practised by myself in 1862 [1] when assistant-surgeon at the European General Hospital, Bombay. A solution of quinine, consisting of twenty-five grains with twenty drops of dilute sulphuric acid in half an ounce of water, was injected into the subcutaneous tissue, well under the skin, of some part of the body (the back of the arm preferred) from a small syringe furnished with screw action. One drachm of such solution may be injected at one time. The operation should be very slowly performed, and the sharpened tube of the syringe be directed in such a manner that gravity may assist in the transmission. It is essential that the quinine be perfectly clear, otherwise deposit of the alkaloid will clog the tube, and, when hardened, it is almost impossible to clear the interior without breaking the instrument. Aitcheson and Scriven advise a solution containing quinine one drachm, tartaric acid half a drachm, distilled water three ounces. In preparing the solution the object should be to dissolve the largest quantity of quinine in a given amount of water by the aid of the smallest quantity of acid; and in this respect samples of quinine differ materially—sometimes a larger, at others a smaller, quantity of quinine than that specified will be dissolved by the same measure of acid and water. If the quinine is perfectly in solution and the acid in proper proportion, just sufficient to dissolve the alkaloid and no more, unpleasant results, as inflammatory action, abscesses, &c., are seldom, if ever, noticed. When I first commenced this practice I did so in

[1] See *Lancet*, 1863; also *Ind. An. of Med. Sci.*, 1863.

most cases presenting. Afterwards I limited subcutaneous injection to those instances when irritable stomach forbids its administration in the usual manner, where the typhoid condition rendered absorption by the mucous membranes slow and uncertain, and when it appeared extraordinarily imperative to give the remedy quickly. I believe quinine acts more powerfully when injected than when introduced into the stomach. But objection is frequently advanced against the slight pain of the trivial puncture.

The difficulty frequently experienced in getting native assistants to use the syringe with care and regularity, and the expense of frequent fracture of a small but costly instrument, led me to limit the use of the subcutaneous injection of quinine to the class of patients above indicated.

In most fevers I believe digitalis, as recommended by Wunderlich, may be advantageously given in combination with baths and quinine. Twenty drops of the tincture, with the same quantity of chloric æther, may be given every three or four hours. This usually has a well-marked effect in strengthening the pulse, lowering the temperature, preventing delirium, and improving the secretions. It is doubtless that we have in digitalis a powerful cardiac stimulant, and it is indicated when there is a rapid pulse and high temperature range. But it must be given with caution, for, notwithstanding the fact of it being a cardiac stimulant, from some unexplained reason it does not act as such when symptoms of cardiac failure have already occurred. It is then contraindicated, but in such cases drop doses of tincture of aconite, given as recommended by Ringer and advised by Pedlow, may be substituted.

Other remedies used as heat-reducing agents are the salicylates, kairin, antipyrin, thallinum, of which I believe the latter may be regarded as the most serviceable, dissolved in alcohol and water. But they all occasionally produce unpleasant effects. Kairin and antipyrin often excite nausea,

sickness, or even stomatitis, and give, especially kairin, a short period of apyrexia. An objection to antipyrin is that it must be used in large quantities. Kairin may also cause pain in the kidneys and blackened greenish urine. The agent may be detected in the urine by NO_5, with which it forms a yellowish reddish ring at the point of junction of two drops placed on a white slab. Thallinum also often produces so much nausea and sickness that it cannot be continued.

As regards complications, sudden weakness of the heart must be met by stimulants, of which I regard port wine as the best, and more will be required by those who are habituated to alcohol. In continued fever, after the first week, especially if the pulse is growing in rapidity and losing strength, port wine or brandy will generally be necessary, as two ounces of the former, or one of the latter, every three hours. But in all fevers the amount of stimulant must be guided by the effects produced. If after stimulants the tongue becomes more moist, or if the skin grows more moist, or if the pulse becomes slower, or the delirium less, the stimulants are doing good; if the reverse occurs, they are doing harm. In severe paroxysmal fevers stimulants are often demanded after the paroxysm, and should be given in combination with good animal broth or farinaceous food, whichever the patient appears to relish best. But in intermittent fever stimulants may also be required in consequence of a prolonged cold stage, which, as formerly observed, sometimes simulates the algide stage of cholera.

Extreme excitability and prolonged sleeplessness may be combated with chloral or morphine. After repeated paroxysms, or when there is restlessness and insomnia, and if the pulse is soft and the skin moist, and when no cerebral determination exists, muriate of morphia in grain doses, or chloral in twenty-grain doses, will frequently produce a tranquillising effect and tend to delay exacerbation. But, with quinine, I am of opinion that the minor or sudorific

calmative effects of narcotics are those from which benefit may be expected. When the full sedative effect is induced they are injurious, excepting in those cases when, the fever having subsided, the exhausted system demands repose. Of the narcotics mentioned I prefer chloral. A combination of opium and quinine, or of morphia and quinine, is often beneficial. The different agents appear to augment each other's powers, and the doses of each may therefore be lessened. I frequently combined the liquor potassæ arsenitis with quinine and laudanum, viz., liq. pot. arsenitis two minims, dil. sulph. acid five minims, tinct. opii eight minims, quinine two to three grains. Before giving an opiate it should be ascertained if the patient does or does not sleep, as he will frequently assert he does not do so when sleeping hours every night. When cerebral symptoms present, the ice-cap may be applied, and if coma supervenes, the cold douche. Convulsions or spasms, as in the cerebro-spinal form of fever, may be benefited by bromide of potassium or chloral, and opium or belladonna liniment may be used to the spine. When the red tongue, tympanitis, and pain denote inflammation of the bowels or peritonitis, frequent doses of chlorate of potash have been strongly advised. Constipation requires castor-oil. Diarrhœa is best controlled with small doses of opium, ipecacuanha, and nux vomica; or, if opium is contraindicated, from ten to twenty minims of turpentine with ten grains of tannic acid may be given in mucilage water. Meteorism is best relieved by a cold compress on the abdomen. Dryness of the tongue is relieved by pieces of ice in the mouth. When hæmorrhage occurs, perfect rest and opium are indicated. If opium is not advisable, owing to cerebral complications, *liquor ferri* may be used. Perforation also requires perfect rest and opium, which offer the only chance. For bronchial and lung complications tonic expectorants are best, and of these ten drops of tincture of benzoin on a lump of sugar may be

given every two or three hours. Lastly, the bladder demands constant attention, and, if necessary, relief by the catheter.

But perhaps more important than any medicinal treatment is good nursing. Early and complete mental and physical rest is a *sine quâ non* in all forms of continued fever, and the more such desideratives can be obtained in the intermittent phase of fever the better for the patient. He should be placed in a well-ventilated room, without bed-curtains or other impediments to the circulation of air. Two beds, to admit of change, are also desirable. The horizontal posture should be maintained, and the patient should not be allowed to exert himself at all. The light should be prevented falling on the patient's eyes, and all noises should be stopped. The bed should not be too soft, and an india-rubber sheet should be placed under the patient. Porcelain utensils are best, and some disinfectant, of which sulphate of iron or carbolic acid are the best, should be placed in the pans, and evacuations should be made lying down. Chlorine gas or other so-called disinfectants may be generated in the room, but free ventilation and an equable temperature are more important. In European climates the temperature of the sick-room should not vary much from 60° Fahr.; in tropical climates it must be warmer, but it should be maintained as equable as possible. The back must be protected against bed-sores by the judicious arrangement of pillows or by air or water cushions, also by sponging daily with concentrated solution of alum. The body should be sponged daily with tepid water, the nurse sponging and drying one part at a time so as to prevent chill from exposure; this relieves the patient and tends to prevent the unpleasant smell so common during fevers. Headache may be relieved by cutting the hair very short and by cooling lotions or ice. Vomiting is often benefited by sucking ice. Cold water may be allowed *ad libitum*, and when the patient is not inclined to drink

the mouth should be frequently moistened with a teaspoonful of water. It very frequently happens that fever patients do not take enough fluid, and it should be the business of the nurse to see that fluid is constantly supplied. Good claret and water may always be given as a drink, or, if preferred, seltzer water, lemonade, or mineral acids with water. When diarrhœa prevails, barley or rice water (congee) is advisable. As regards diet, too great abstinence is only worse than overfeeding. Proteine compounds are, however, interdicted, and carbohydrates more required, such as oatmeal, gruel, and meat broths. When the enteric phase of fever prevails with its intestinal complications, milk *ad libitum*, beef-tea, broths, jellies, extract of beef, Liebig's raw-meat soup, should be the only articles of diet, and no solid food should be allowed under six weeks or two months. Eating an orange or a piece of potato, or even drinking an effervescent draught, may cause distension and rupture of the healing bowel. During convalence from any fever quinine and mineral acids will be advisable.

MASKED MALARIAL FEVER.—This condition I formerly ventured to describe as masked malarious fever, and as one which had not been hitherto fully recognised by writers on tropical diseases.[1] But the correctness of applying the term fever to a condition in which the pulse is not always accelerated was questioned at the time. It was contended that there could be no fever without some such excitement, and that the term was therefore improperly used. But the researches of Parkes, Jones, Virchow, and Wunderlich have demonstrated that the febrile condition is associated with more abundant excreta from the body than in health. The bodily temperature and the amount of excretions bear some relation to each other, but both these may be altered without any sensible variation in the force or frequency of the pulse, as judged of by the fingers, although the sphygmo-

[1] *Bombay Med. and Phy. Trans.*, 1861; *Ind. An. Med. Sci.*, 1866.

graph may detect variations at an earlier period. The metamorphosis of tissue or the change in the amount of the excreta may not be sufficient to affect the pulse, as judged of by the fingers, which will only become excited when the changes going forward in the system become more rapid. The febrile state exists without the ultimate results of a very high temperature, cerebral affections, &c., and there is no doubt it may be present in a still less intensity—not sufficient even to alter the characteristics of the pulse, as judged by the touch. Increased excreta being the first step, increased temperature—evidenced in masked fever by burning palms and heat in the soles of the feet—is a second link in the chain, but alterations of the pulse sufficient to be discovered by the touch belong to a third or more remote place in the progress of events.

Masked fever may be regarded as a minor manifestation in the system of that combination of the operation of the various conditions of climate and circumstances by which we are surrounded. Any cause reducing the system below *par* will add to the sum total of climatic and local circumstances, especially the scorbutic or syphilitic taint, hæmorrhoidal discharges, prolonged lactation, leucorrhœa, &c. The individual affected complains chiefly of the heat, dryness, and burning in the palms of the hands, less frequently in the soles of the feet, which may be persistent with slight remissions when the parts become a little moist, or there may be distinct intermissions of days or weeks. There is more or less general uneasiness or lassitude, perhaps slight headache, especially in the morning, but no decided pain anywhere. The pulse is not excited, but weakness may be perceived. The skin, excepting of the palms or soles, does not feel warm, but the thermometer will show that the temperature is slightly higher than natural. The appetite generally remains good. Sleep is restless and disturbed by dreams. The alvine evacuations are normal, but the urine

often shows excess of urates. Sometimes, in addition to the burning of the palms and soles, there is heat about the cheeks and eyes, and occasionally injected conjunctivæ. It is, however, questionable if this latter condition is not more due to the rubbing the eyes which the burning and heat induce rather than to the same cause as the burning itself. The person may also be annoyed by occasional periodic beating, or singing in the ears, as of a kettle boiling or distant crickets chirping—more particularly noticed during the silence of night. I have also observed frequent micturition, especially at night (the sign of an irritable bladder), is often present, and when this is the case the possibility of incipient disease of the kidneys should be recollected. Persons affected with masked fever are generally disposed to chilliness, and require a large amount of cold-weather clothing. On the other hand, they are soon depressed by heat. This condition may prevail for months or even years, and is often so slight as scarcely to attract much attention, but in other instances it constitutes a perpetual source of annoyance and discomfort.

By a comparison of the symptoms here given with those of anæmia, leucocythæmia, &c., it will be observed that the conditions are essentially different. Although masked fever may be the first link in the chain of deterioration ending in anæmia, the latter is something more than the former. In masked fever the *materies morbi* appears to be excreted from the system in sufficient quantity to prevent decided blood-deterioration; in the latter it accumulates in excess, and hence those secondary effects on various organs referred to under anæmia. (p. 28.)

It not unfrequently happens that masked fever is accompanied and complicated by cutaneous affections, of which eczematous eruptions are the most frequent. The irritation thus produced materially adds to the general irritability, and renders the febrile condition more marked. There

is also a considerable tendency towards supra-orbital neuralgia.

The treatment consists in an essentially tonic and somewhat stimulating régime, with nourishing diet. I have frequently observed that persons thus affected consume a large quantity of meat, and within moderate bounds the inclination may be followed. A daily proportion of good sound ale I would advise in preference to wine or spirits. Coffee has long enjoyed a questionable reputation as an antiperiodic, and has appeared to me to exert some beneficial influence. Delioux stated that roasted coffee is far from possessing powers equal to those of the raw berry, and advises an infusion in cases of malarious cachexia. As regards medicine, I have more confidence in the *liquor potassæ arsenitis* than in other reputed febrifuges. Change of climate is, however, the only certain means of cure. Simple change of *locality* will not suffice to overcome the malady. The removal must be into a northern European country. The Indian hill ranges do not afford permanent relief. And in England the burning palms and singing in the ears will frequently for months demonstrate that even the British climate will eradicate the condition only after a prolonged residence. Probably few medical men would consider it necessary to recommend their patients a radical change of climate when presenting merely the symptoms of masked fever, and most patients would object to taking this step on account of an ailment apparently so trivial. It cannot, however, be forgotten that this condition is the first link in a chain of events which will probably ultimately render removal imperative.

A condition somewhat resembling masked fever sometimes arises apparently from saturation of the system with quinine or arsenic given for the cure of fever. The blood may be said to be poisoned with these agents. When, therefore, masked fever occurs in persons who have taken much

quinine, the possibility of such being the case should not be forgotten. Mild aperients and diuretics are then advisable.

Prophylaxis.—The protection against fevers of all descriptions is good sanitary surroundings and good personal hygiene, but when these terms are analysed it will be seen that they resolve themselves into protection of the person from chills. Under personal hygiene is clothing, of which the best is flannel, the superiority of which texture in defending from chill is admitted. Also nutritious food, poorly fed people being most likely to be affected by vicissitudes of temperature. And it is among the poorly clad, the poorly housed, and the poorly fed that fevers of all kinds are most destructive. Care also against exposure to the sun, which renders the skin so liable to after-chill. The prophylactic issue of small doses of quinine with a view to its tonic and stimulating effect on the nerves, whereby the system is braced against changes of temperature. Klebs recommends the juice of a lemon twice a day, which probably acts by counteracting any scurvy taint which would otherwise favour susceptibility to chill, as indeed all debilitating agencies do. Among good sanitary surroundings the first desideratum is good drainage, with consequent freedom from damp and its effect, chill. Good conservancy and cleanliness generally prevent that vitiation of the atmosphere which leads to contamination of the blood, and consequent further susceptibility to changes of temperature.

CHAPTER XVII.

FUNGUS-FOOT DISEASE OF INDIA.

Synonyms: *Madura foot. Mycetoma. Morbus tuberculosis pedis. Ulcus grave. Podelkoma.*

THIS disease does not appear to have been recognised beyond the limits of Hindostan, where it was first described as a distinct malady by Godfrey of Madras, in 1846.[1] It attacks natives, both male and female, no cases in Europeans or half-castes having been recorded. The peasantry or cultivators appear more subject to the disease than other classes, and it is more frequent in Western India than in Bengal. Although supposed to originate from a vegetable fungus, the nature of the soil does not influence its prevalence, as it is frequently met with on black cotton soils, and on sandy semi-desert tracts, as in Western Rajpootana. The fact of its occurrence in these sandy wastes, where the rainfall scarcely averages seven inches annually and where water is hundreds of feet from the surface, is important, as if there is a fungus connected with the disease, it must be one capable of flourishing not only in moist localities, but also in extraordinarily dry places. The foot is the part most frequently attacked, but I have treated it in the hand, in the calf of the leg, and on the thigh.

The commencement and progress of the malady is usually very slow. I have had persons apply for relief after suffer-

[1] *Lancet*, 1846.

ing from it for years, and cases have been reported where it has existed for thirty and forty years. The history of a case is that of gradual swelling of the part affected, with eventual redness, or rather in the native duskiness of the skin, underneath which black slightly nodulated marks usually appear, as if gunpowder or Indian ink had been pricked into the integument. Eventually the integument gives way, and fistulous orifices form. When fully developed the part presents appearances not unlike on a cursory view what is observed in some forms of caries of scrofulous origin, and it was described years back by Mr. Eyre as tubercular disease of the foot. The foot is increased in circumference, the enlargement seldom extending beyond the ankle, and openings are scattered over the affected part, from which pus and black granular material may ooze. The foot affected is prone to run in a line with the leg, and to become everted or inverted. When the hand is affected there is similar swelling, and the wrist is shortened owing to destruction of the metacarpus or even of the carpus, and the consequent irregular tension of the extensors and flexor tendons.

A section of a diseased part shows the bones to be very much softened and enlarged, and more or less destroyed by a series of sharply-defined cavities; some quite isolated, but the majority communicating by a series of complex channels, the whole containing glairy fluid and solid concretions. Both cavities and channels are lined with a dense glistening membrane, composed of white fibrous material, and the surrounding tissues have been found in a fatty condition. The idea has been suggested that some entozoon must have been at work to produce the channels which riddle the foot, and so the malady has been attributed to the guinea-worm, which is known sometimes to die and become calcareous in the body.

V. Carter believed the matter formed in the cavities to be a fungus, and in this Mr. Berkley agreed, naming it

chionyphe Carteri, and the disease has been divided by Carter[1] and others into several varieties. *First*, the pale or ochroid, from the discharge, which consists of whitish-yellow or pink roe-like bodies about the size of millet seeds, or of grains of cayenne pepper. *Secondly*, the dark or melanoid form, from the dark brown or black granular bodies not unlike grains of gunpowder which escape through the sinuses, and which may be found in the interior, of all sizes to that of a walnut, tightly fitting into a cavity or surrounded by gelatinous matter. The yellow roe-like particles are composed of a nucleus of granular waxy consistence. The red particles consist of phosphates and carbonates with traces of iron. The dark masses are lighter internally, and present a radiating structure, and contain cells surrounded by filaments of apparently fungoid nature, which, however, cannot be cultivated. The occurrence of these fungoid-like filaments in the dark variety has caused some to regard them as the essential cause, but the light variety shows an absence of fungoid filaments. It appears as if both forms were due to the gradual transformations of fat (Lewis and Cunningham).

Admitting the presence of a fungus element in this disease, much difference of opinion has been expressed regarding the manner in which the germ enters the system. That it does not gain entrance through the skin appears probably because the malady always commences internally, and I do not think it could be diagnosed as fungus-foot when swelling only is present, and the black material has not appeared mottling the surface. If the entrance were effected through the integument we might expect to find external swelling and black matters first, instead of deeper-seated affection. Lewis and Cunningham's theory is that the germs may enter through the intestinal canal, becoming accidental complications in a deep-seated abscess, which affords a suitable nidus for their growth. There are, however, other reasons to

[1] *Mycetoma, or the Fungus Disease of India*, 1874.

believe the superficial parts are first affected. In four cases I incised the integument, and cut and scraped away the diseased parts.[1] In three of these cases the cure was permanent eight months afterwards, but the result of the fourth case was not ascertained. Drs. Harvy and Spencer treated cases successfully at Boulpoor in the same manner, and Dr. Eddowes [2] at Erinpoora cured by excision and the use of caustic potash or nitric acid.

The question indeed may be even now asked whether there be any fungoid germ at all. There certainly is not in the white variety of the disease, and the fungus, if such it be, found in the dark varieties may be accidental. I believe that whether the internal appearances are white, reddish, or black, it is the same disease in different stages, and I am not prepared to assert that the malady is not consequent on the presence of a dracunculus (or its ova) in the parts, which has undergone changes causing it to act as a foreign body, the blackened material being pigment, perhaps from extravasated blood.

Treatment.—There is no cure for this disease excepting, when superficial, cutting down upon and removing the diseased parts, and in other more severe or prolonged instances amputation. Bleeding has sometimes occurred after amputation for this disease from the end of the divided bone.

[1] *Ind. Med. Gaz.*, Nov. 1867. [2] *Ibid.*, Sept. 1867.

CHAPTER XVIII.

GOITRE.

Hin.: *Geega.*

GOITRE has been described under the terms adenoid, cystic, fibrous, vascular, and carcinomatous. The vascular or fibrous varieties are most frequently seen in India. It is very prevalent in some parts of India, especially in the Terai, where one in ten are afflicted; it is also more or less prevalent throughout Oude, and in the Himalayas and other mountainous districts. It also prevails at the base of and among lofty mountains in other parts of the globe, especially in Switzerland. Mountains, however, are not essential to its prevalence, as it occurs in Tirhoot, hundreds of miles from mountains, and it is frequently seen on the sandy plains of the semi-desert districts of Western India. Females are most liable, but it affects both sexes, and presents at all ages. Europeans in India are comparatively seldom affected. Animals—as goats, kids, sheep, lambs, dogs—are also liable. The malady has been attributed to numerous causes, as elevation, valleys, snow-water, particular food, changes of temperature, intermarriage, iron pyrites, and in India to *reh* or *ossur* (a saline efflorescence from the ground composed of sulphate of soda in alliance with potash, lime, magnesia and silica) which appears on undrained soil in many parts of the country. It has also been attributed to malaria, and Fayrer is under the impression that the causes which produce anæmia and splenic maladies are concerned in bronchocele; but on the other hand it is comparatively seldom

found allied with anæmia. Laborious exertions in constrained positions and carrying loads on the head have been credited as a cause. It has always been supposed to be hereditary. But the general view is that it is connected with lime or magnesia in the drinking water; and it has been stated that, however differently it presents itself, there is one unvarying element in the presence of lime in the drinking waters, and which is to be found in the local geological strata, or in such a position that it can be washed down with the water consumed. Moreover, the disease has been known to affect a family who, after using surface water, had a well sunk in limestone rock (Fayrer). It has been theorised that the quantity of lime taken into the system diminishes the calibre of the pramnia in the base of the skull, interfering with circulation and thus becoming instrumental in the production of bronchocele and cretinism. There are, however, objections to attributing goitre to any of the causes named. Those who live on elevated localities in many parts of the world, on the Neilgherries and Mount Aboo for instance in India, do not suffer from the disease, and the same remark applies to valleys. In Greenland and Lapland, where snow-water is commonly used, there is no goitre, while it is common in Sumatra, and parts of India where snow is unknown. There is no evidence to show that any kind of food produces the disease, for it prevails among populations living on the most diverse diets. Changes of temperature, although greatest in hilly districts, prevail everywhere, and it has already been stated that in some mountainous localities the disease does not present. As regards intermarriage this occurs quite as often among classes where goitre does not occur as where it is found. As regards its origin from iron pyrites, reh or ossur, there is not a particle of evidence supporting such a view. Malaria has been credited with the production of so many ailments and not proved to excite any, that it may be dismissed from consideration, even if we did not know that goitre prevails

in Switzerland, which is not a malarious country, if it is to be judged, as other countries are, by the presence or absence of malarious fevers. Moreover, although goitre is sometimes associated with cachexia, the subjects of the malady are very often otherwise healthy, presenting no symptoms of malarious degeneration. If active and laborious work in constrained positions were a cause, it would occur in other localities, for occupations of the kind are not confined to goitrous districts. It has been sufficiently proved that although some instances may appear hereditary, the majority are not so (Macnamara). With respect to lime as a cause, the evidence is far from conclusive. It is stated that the districts round Dhurmsala, where goitre prevails, are free from lime, magnesia, or iron. H. N. Macnamara [1] states the facts in the history of the disease are quite unexplainable on the lime or magnesia theory. For example, goitre is not endemic in Southern India, where every variety of water is found. Chevers seems inclined to attribute goitre to damp, as he states it is prevalent on white damp *bhat* soils not requiring irrigation, but unknown on reddish sandy dry *bangar* soils; but, as previously mentioned, it occurs on sand in Western India. All that we can certainly say is, that where goitre prevails, as a rule, lime will be found in the water; although the rule is not of general application, that goitre is most prevalent in valleys at the bases of, or within the spurs of, or near lofty mountains; and that those who come down into the valleys, especially immediately after the rainy season, are most likely to be attacked.

Goitre may come on almost imperceptibly, but it often presents very quickly, which has been accounted for by the extreme vascularity of the thyroid gland, and the size of its arteries. McLelland noted that the tumour does not always originate in the thyroid gland, but sometimes lower down, a statement supported by Chevers. When it attains any size,

[1] *Himalayan India.*

it causes great distress by pressing on the trachea and nerves and vessels of the part, resulting in difficulty of breathing and of swallowing, headache, and change in the tone of the voice, which becomes reduced in volume, and there is sometimes diminished muscular power on one, or even both sides of the body. Occasionally, paroxysms of dyspnœa occur, or there may even be acute asphyxia. It is noted that the size of the tumour usually increases during the catamenial period, and flooding in childbirth of goitrous women is common. Goitre is not associated in India with cretinism as it is in Switzerland, the latter appearing to be an hereditary affection, which goitre is not, and the connection therefore, although frequent, seems to be accidental. Neither is Indian goitre associated with exophthalmos excepting in very occasional instances.

Treatment.—Captain Cunningham and Major Holmes, when stationed in the Terai in 1854-55, treated 25,000 goitrous persons with great success. The recipe they used is, 'Melt three pounds of lard or mutton fat, strain and clean; add nine drachms of biniodide of mercury, well powdered; work up well, and keep it from the rays of the sun. About an hour after sunrise, apply and rub in for ten minutes, the patient sitting in the sun and holding his goitre towards it. At noon, there will be pain from the blistering effects, but no pustules. Apply carefully at 2 P.M., and let it be gradually absorbed.' Now, notwithstanding the success attained by the gentlemen named, I failed when I tried this measure years back on its being first announced; and Fayrer says it often produced inflammation of the neck and salivation. Coates states of his patients thus treated, 'one third were cured, one third benefited, and the remaining third not relieved.' I think that a biniodide ointment half the strength of the above, with iodide of potassium internally, accompanied by tonics if necessary, is the best method of delaying the progress of the disease. Removal to another

district is, however, a more certain means of relief than any kind of medicine. Operative interference is rarely justifiable, as the dangers from hæmorrhage, wounds of the trachea, wounds of large nerve trunks, and entrance of air into veins, are great. Fayrer ligatured the arteries, but, beyond temporary relief, did no good. Setons are equally useless.

CHAPTER XIX.

HYDROPHOBIA.

When the large number of pariah dogs in Indian villages, and the great influence of heat in exciting canine madness are recollected, it ceases to be matter of surprise that hydrophobia is frequently seen in the East. And this notwithstanding that the fact of heat conducing to canine madness, and the fact of hydrophobia being more prevalent in the East than in the West, have been questioned. The great majority of the cases arise from dog-bite, and most of the remainder from cats; a few arise from the bites of jackals, foxes, camels, and wolves. Saliva from the mouth of a rabid animal undoubtedly contains the poisonous agent. No other secretion has been discovered to be poisonous. Advancing decomposition is said to destroy the activity of the venom, but dried saliva will maintain its virulence for a lengthened period. Although a bite is the most usual manner in which the poison is transferred, it may be conveyed, when there is the slightest abrasion of the skin, by a lick from a rabid animal. A healthy dog has communicated the disease by a bite given immediately after it had been fighting with a mad dog, the saliva from which was no doubt hanging about its jaws. It has also resulted from the teeth being used to loosen a knot in a rope with which a mad dog has been tied, whether there was abrasion was not ascertained. But Youatt and Portall considered the saliva of a rabid animal could not touch a mucous membrane without danger.

It has also been asserted that contact with a rabid dog's saliva when there is no wound or abrasion will cause the disease. Dr. Watson mentions an instance where the tooth of the dog merely indented the man's hand, and papers published by Mr. Hutchinson some years ago tend to prove that contact without wound or abrasion may occasionally engender the disease. Such instances must, however, be received with caution, for there is always the possibility of a minute abrasion having existed. Still, as the syphilitic poison will act, not only on the semi-mucous membrane of the glans and prepuce, but also on the integument of the penis, when there is no visible abrasion, so the same may be the case with the hydrophobia poison. It has been asserted by Monsieur Putegnat and others, that hydrophobia may arise from the bite of a simply enraged animal, especially if the sexual appetite existed at the time. Further, it has also been stated that the malady may arise from the bite of a healthy dog. Several instances are on record where the man died and the dog remained well. These must have been, I think, cases of the spurious hydrophobia afterwards mentioned. Other cases are on record in which the disease followed the bite of a dog which did not present symptoms of madness for several months afterwards. It is therefore possible that rabies may affect a dog as a mild and insignificant, not recognisable malady, or that the disease may be latent in the dog as it is in the man, yet the dog may be able to convey it.

That a very slight scratch or wound, whether inflicted by the teeth or claws on which saliva may have fallen, is sufficient to induce the disease, there is abundant evidence in the writings of Youatt, Breschet, Majendie, &c. But it does not follow that all persons injured by a mad dog get hydrophobia. All persons are not perhaps equally susceptible, or the quality of the poison may vary, or it may be deposited on clothing. It has been computed that only one in twenty

of those bitten by mad dogs suffer. A case is mentioned by Hunter of twenty-one persons being bitten by the same mad dog, of whom only one suffered. Froillet gives another instance, when seventeen were bitten, and only one suffered. Dolan states that when no preventive measures are used at least half escape.

The latent period of hydrophobia is very variously stated. Watson says the majority of cases occur between five weeks and eighteen months,[1] but the affection may present three or four days after a bite,[2] or not until three[3] or even twelve years have passed away.[4] But one of the most recent authorities[5] states that incubation is rarely less than a month, that the shortest case on record is twelve days, the average being from six to seven months, although it may occur as long as ten years afterwards. As the poison of syphilis or malaria remains in the system for an unlimited period, hydrophobia poison may do the same. It is as difficult to explain an incubation of six months as of six years.

Males and children suffer most, for obvious reasons, but females are quite as liable as males if bitten by a rabid animal.

Symptoms.—A wound inflicted by a rabid animal heals completely as an ordinary wound would, and there are usually no symptoms during incubation, unless in circumstances where fear of the disease exists, when there is mental depression and anxiety, vague feelings of uneasiness, gloom, irritability, frightful dreams, and sometimes twitching of the muscles of the face. Persons thus suffering from dread of hydrophobia have been known to commit suicide. In some cases, a slight pain or uneasiness of the wounded part has been observed. In a still smaller number of cases it becomes

[1] Watson's *Lectures*, vol. i. [2] Pickell, *San. Rev.*, No. 12.
[3] Case by the author, *Trans. Bom. Med. and Phy. Soc.*
[4] Pickell, *San. Rev.*, No. 42. [5] Dolan *On Hydrophobia.*

red, or even swollen and sometimes suppurates.[1] Numbness and stiffness of the affected limb have also been occasionally observed. Cases commencing suddenly with rigors have also been reported. But in the majority of instances, the first evidences of the disease are malaise, irritability, low spirits, disturbed sleep, alternate chills and heats, with some discomfort or stiffness about the throat, and slight difficulty of swallowing. This soon increases gradually into spasm, involving the muscles of respiration and causing first a catch in the breath. Or, the discomfort about the throat may suddenly pass into suffocative spasms, most probably on some occasion when the person attempts to drink. There is no sub-maxillary, parotid, or cervical enlargement in connection with the condition of the throat. Whether the spasms commence suddenly or gradually, they recur after variable intervals of time, from minutes to hours, involving first the extraordinary muscles of respiration, and eventually the whole muscular system. The face is blue and turgid, the eyeballs protruding, the patient foams at the mouth, and claws at the throat, as if to move some obstruction, and priapism has been often noted. These general spasmodic seizures, although resembling those of tetanus, are succeeded by perfect intervals of relaxation. Between the spasms, abundant viscid saliva, which cannot be swallowed, collects about the mouth, occasioning perpetual hawking and spitting. At first these spasmodic attacks are excited only by the attempt to swallow fluids; afterwards, the sound or sight of fluids, any motion near the sufferer, even a draught of air, or a look from a bystander, or the movements of spontaneous deglutition, are sufficient to excite them. Aversion to anything white has frequently been noticed, and the sight of a dog has been known to bring on the spasms when the sufferer was not aware of the nature of the disease. As the respiratory spasm spreads to wide convulsions, so the mental distress proceeds

[1] Peet, *Bom. Med. and Phy. Soc. Trans.*

to frenzy, especially during the paroxysm. Sometimes the patient rushes wildly about the department in a state of maniacal fury, staring fiercely, and clawing at the throat. It has also been reported that he sometimes barks like a dog (for which probably the hawking has been mistaken), and that he tries to bite his attendants (for which probably the spasmodic movements of the jaw have been mistaken). Sometimes, during the intervals, he is remarkably silent, and refuses to answer questions; at others, there is a long and garrulous talking, although the mind often remains unclouded to the last. In the intervals between the paroxysms the patient is quiet and reasonable, but constantly endeavouring by hawking and spitting to clear his throat. Sometimes there is little or no febrile action; at others, the temperature rises two or three degrees, with hot, dry skin, parched tongue and thirst. Towards the end, the surface is covered with cold perspiration. The urine often contains albumen, and sometimes sugar, but is frequently natural. In some cases a deceitful calm occurs before dissolution, the special symptoms remitting or disappearing altogether. After which, convulsive paroxysms occur with increasing rapidity and violence, in one of which the patient dies asphyxiated from spasm of the glottis; or he may sink exhausted at an earlier period from cardiac failure. The termination of a recent case is thus reported by Surgeon-Major Keegan, 'Whilst talking, a deadly hue came over his countenance, he lost consciousness, the heart's action failed, and he suddenly expired.' Pain in the part bitten has been known to precede death.

It is seldom all these signs and symptoms are fully manifested. Although most observers agree in the main phenomena of dread of liquids and of air, secretion of viscid mucus, and hawking and spitting, with paroxysmal attacks of a spasmodic nature, still the variety in minor points may be such that in this respect hydrophobia, as has been said of

other diseases, may be truly designated a 'Protean' malady. Cases have occurred in which fear of, and spasm from drinking water, were not very strongly marked, and ceased altogether in the later stages.[1] In other instances the patient could swallow when the eyes were shut, and this should always be tried.

Acute endocarditis with mitral systolic murmurs have been noted as a complication, there having been no previous attacks of rheumatism. Cardiac affections of the kind have even been stated to have been mistaken for hydrophobia, and *vice versâ*. It has been stated by Marochelli that at the commencement, or before the development of the disease, vesicles rise under the tongue, which have been thought distinctive of hydrophobia as other eruptions are of other diseases. This is said to have occurred as early as three days after a bite, and may be looked for. I have never seen it, and Dolan says the appearance has not been confirmed.

Duration.—The ordinary duration of hydrophobia is from one to four days, but the case is sometimes extended to six or eight days. In rare cases of recovery more or less spasm about the throat may remain for an indefinite period.

Diagnosis.—The eighth pair of nerves are chiefly and primarily affected, and consequently the spasmodic muscular action is at first confined, and sometimes altogether confined, to the neck, larynx, and pharynx. The symptom of greatest diagnostic value is therefore the respiratory spasm excited by attempts to swallow. The continued hawking and spitting with aversion to fluids are also of great value as diagnostic marks. In tetanus, with which the disease may be confounded, there is no fear at the sight of liquids, none of the hawking and spitting of hydrophobia, no paroxysmal respiration, spasm, and an absence of sensorial or mental

[1] *Mad. Med. Jour.*, No. 13.

disturbance. Moreover, in hydrophobia there is no trismus as in tetanus. The convulsive spasms in tetanus are continuous or tonic, while in hydrophobia there is complete relaxation after the convulsive attacks. Lastly, there is generally a history of dog-bite in the one case and not in the other, but probably the mark of some other injury. Still, so great are the variations from the type in both maladies that the diagnosis may be obscure, especially if there is no prior history. This obscurity is sufficiently explained, if it is correct, as Dolan states, that there is a *tetanoid* form of hydrophobia, in which general spasms occur early; but even then they intermit, are excited by attempts at deglutition, and there is no trismus. In my experience tetanus has been more likely to be mistaken for hydrophobia than the reverse.

Although, according to Hecker, hydrophobia existed at least 400 B.C., it has been denied that there is any such disease. Thus, years back Sir Isaac Pennington denied that hydrophobia existed at all, and Garard in 1827 asserted there was no specific poison; both these authors attributing the symptoms arising after the bite of a dog to mental excitement or fear. Only recently Dr. Dulles asserted it is not a specific disease arising from the bite of a mad dog. Dolan also admits that mental excitement may terminate in a spurious hydrophobia presenting symptoms somewhat resembling the genuine disease. After the bite of a dog spasms are felt in the throat, but they are not of the respiratory character of hydrophobia; it is mere dysphagia, and an effort overcomes it. This he regards as a distinct nervous affection, or as Trosseau termed it, 'mental hydrophobia.' As showing that mental force has much to do with hydrophobia, cases may be referred to of persons who, commencing to suffer from hydrophobia, have by force of will gone through great exertion, exciting profuse perspiration, with the result of the disappearance of the symptoms.

Inflammation of the œsophagus has given rise to symptoms somewhat resembling hydrophobia, and the difficulty of swallowing occurring in pericarditis, and other cardiac ailments, have been mentioned by French authors in a similar manner. But endocarditis, as previously referred to, has been noted as a complication of hydrophobia. Again, a type of intermittent, from its presenting peculiar symptoms, has been termed 'intermittent febris hydrophobiæ.'[1]

Hysteria, among other of its vagaries, occasionally mimics hydrophobia. I have in recollection a case in which the symptoms were sometimes those of hydrophobia and sometimes those of tetanus, the young woman affected never probably having heard of either disease. The history of the case, the age, sex, the globus, and other hysterical phenomena cannot fail in establishing a correct diagnosis.

The *prognosis* is most favourable the longer the spasms remain limited, and less so when there are general convulsions, or when the suffocation spasm of the glottis is prominent. Mental disturbance and signs of exhaustion warrant the expectation of a fatal termination.

Post-mortem appearances.—Dolan states the phenomena of rabies, as evinced by morbid anatomy, depend on structural alterations in the medulla oblongata and spinal cord, influenced by a specific virus. Pasteur asserts that the spinal cord is affected before the medulla, and that the virus is contained in the nerves of the body, and in the salivary glands. A post-mortem usually displays general fluidity of the blood, with sometimes congestion and serous effusion between the membranes of the brain and spinal cord. Blood has also been found extravasated in the cervical portion of the latter. The fauces, tonsils, root of the tongue, œsophagus, trachea, and bronchi have been found reddened, congested, and vascular. The salivary glands have been found reddened, enlarged, and vascular. The

[1] *New York Med. Jour.*, Dec. 1883.

lungs are sometimes congested. The eighth pair of cerebral nerves and the cervical sympathetic have been found purplish. Traces of hydrocyanic acid have been found in the blood. There are also microscopic evidences of infiltration in the salivary glands, and of minute changes in the nerve-centres. They consist in the accumulation of leucocytes around the vessels, and their infiltration into the adjacent tissue in the region of the medulla which is contiguous to the lower part of the fourth ventricle, *i.e.*, the neighbourhood of the respiratory tract. The only change in the nerve-elements themselves is a granular degeneration of the ganglion cells in the region affected. In dogs similar changes are found (Dolan). It is theorised that by the infiltration of leucocytes tissue may be broken up, and what are practically minute points of suppuration may result. But occasionally there is complete absence of any recognisable organic lesion.

Treatment.—Some few cases of recovery have been recorded[1] which appeared to be due to the action of remedies. Other cases have terminated favourably, apparently in spite of remedies. Most of these favourably terminating instances have commenced to mend at an early stage of the disease. It is uncertain if any medicine really exerts the least influence. Pages might be filled with an account of the extraordinary means which have been resorted to. Among other curious recipes the blood of a spoon-bill duck was lauded by Dr. Mayer, St. Petersburg, so lately as 1828. Jean Baptiste Chornel admitted no less than 88 plants as specifics against hydrophobia. In an old work entitled 'Strictures on the Present Practice of Physic,' 1758, the more rational plan of salt and water to wash the wound, and salt and water internally is urgently recommended.

Avicenna wrote, 'Cura propinqua est, ante terrorem aquæ,' and Baungmarten, a German surgeon states, 'Before

[1] One by the author, *Bom. Med. Phy. Trans.*, 1861.

the dread of water sets in, the cure is not only practicable but not unfrequent.' Two or three cases of recovery are on record after the use of vapour baths. Similar recoveries have been reported after the use of curara, of which there is a tolerance in this disease, one grain having been used instead of the ordinary dose of $\frac{1}{11}$ to $\frac{1}{3}$ of a grain. Surgeon-Major Roy has recently revived the proposal of inoculation with snake-venom on the principle of the two poisons being antagonistic. Then there is M. Buisson's treatment. The wound is washed with a solution of ammonia, the patient has a daily vapour bath at a temperature of 42° to 48° R., with a view to producing profuse perspiration for seven consecutive days, and draughts of a hot infusion of borage, also with a view to promoting diaphoresis. Dr. Buisson is said to have cured himself after the development of the symptoms of hydrophobia. He regards the method as an infallible preventive of hydrophobia, and as an equally certain cure if begun on the first day of the developing disease; on the second day its effects are less certain, and later on it has no effect in staying the malady.

Notwithstanding all this the established practice of the present day attempts little more than the relief of the sufferer by opium, chloroform, and other sedatives or narcotics. Hydrate of chloral with bromide of potassium has sometimes appeared useful. A 5 per cent. solution of cocaine applied to the back of the tongue and fauces has been known to check the spasms. Ice-bags to the spine; a darkened room; a bed surrounded by light curtains; to relieve the hyperæsthesia of the surface by protection from draughts, are desirable adjuvants. Tranquillity and nourishment are perhaps of the most importance, with effectual, but as little as possible irksome restraint.

I have treated two cases successfully[1] by alternate cold

[1] *Trans. Bomb. Med. and Phy. Soc.*, 1860–61. 'Marwar, The Land of Death,' *Ind. An. Med. Sci.*

affusion and chloroform, the throat and upper part of the spine being also vesicated with nitrate of silver. At first the spasms appeared increased by the cold water, but afterwards there was a sedative effect, which I think as it were paved the way for the chloroform which would otherwise have congested the brain.

As one of the principal characteristics of the disease is spasmodic closure of the glottis, Mayo and Marshall Hall long since suggested bronchotomy or tracheotomy, and successful cases have been recorded after this operation.

Pasteur seems to have recently proved that inoculation renders dogs proof against the disease after they have been bitten by mad dogs, and similar success has been recorded by the same authority in the human subject. But we cannot yet regard this as an established method of either prevention or cure. The sources of fallacy are various. We do not know the proportion of individuals bitten by a mad dog who become the subjects of hydrophobia. It does not follow that a person so bitten will get hydrophobia, and before a cure is asserted it must be proved the person would have had hydrophobia, or success must be invariable. Inoculation for rabies appears a plausible theory, but it remains to be seen if it can be established as an art, or if it will not share the same oblivion as inoculation for syphilis. So far back as 1849 the efficacy of inoculation with rabies-poison was discussed in Jahr's 'Klinischen Anweisungen,' and Valli of Tuscany, and Bering of Philadelphia are mentioned as having used this means ineffectually.

The saliva of persons with hydrophobia has been proved capable of communicating the disease to animals (Dolan); therefore attendants should be careful not to let it come in contact with abrasions. As a matter of precaution it is well for attendants to wash the face, and particularly the eyes, frequently with carbolic acid lotion. Although there is no instance on record of the disease having been communi-

cated to an attendant, Dolan advises that if an attendant is injured by a patient, the injury should be treated as the bite of a rabid animal.

Treatment of the Bite of a Rabid Animal.

There is reason to suppose the virus remains dormant near the wound, and only becomes absorbed at the period of recrudescence—not, like the serpent's venom, being absorbed immediately. Hence the greater amount of benefit which may be expected from excising the bitten part, as recommended from the time of Galen downwards. When the leg or arm is bitten a ligature should be tightly tied round the limb, the wound should be allowed to bleed, and the person or some one else should forcibly suck the wound. Provided there is no wound on the lips or mouth of the person sucking, experience shows that only infinitesimal danger can be incurred, but as a matter of precaution the mouth may be rinsed with vinegar and water. Then as soon as possible the part should be cut out, and water-dressing applied. If position forbids cutting the part out, several punctures should be made near, and bleeding encouraged by suction with cupping glasses and hot water. If the bitten part is not excised at the time, it should be effected days or even weeks afterwards should the person appear fearful of hydrophobia. But when the patient is not seen until the wound is healing, it will be better, in the absence of fear and in the absence of symptoms, to leave it alone. Dolan recommends after ligature and sucking, nitrate of silver as an immediate application, and carbolic acid, or nitric acid, if any time has elapsed; but the treatment, as above, seems more likely to meet the exigencies of the position. It is worthy of remark that Youatt asserted he had applied lunar caustic to 400 persons bitten by dogs, and four times on himself after bites by dogs decidedly rabid, and hydrophobia did not appear in any one instance. As the action of lunar caustic is very superficial, and as Pasteur asserts inoculation will not only lengthen the stage of incubation, but also protect from the disease, it may be that Youatt was so protected. However this may be, I should not feel disposed to trust to either inoculation or nitrate of silver.

But if after a bite from a mad dog the person will not submit to the knife, or if a sharp knife is not at hand, nitrate of silver may be thoroughly applied; or if not available, strong nitric or sulphuric acid, or caustic potash, or boiling oil may be applied to the wound. Or the actual cautery may be used in the shape of a red-hot iron wire. It is stated that the pain of the actual cautery is not nearly so acute as believed, especially if the iron is very hot, and it is pressed firmly into the wound. Filling the wound with gunpowder and flashing it has been recommended in the absence of other means; also rubbing the wound with gunpowder

after scarification, but this leaves an ugly mark afterwards. Personally, I have more reliance in free bleeding to carry away virus than in its destruction by an escharotic agent afterwards. In all cases everything should be done to reassure the patient, and he should be made to understand that everything has been done which can be effected.

CHAPTER XX.

INSOLATION.

Synonyms: *Coup de soleil*; sunstroke; *ictus solis*; heat apoplexy; hea asphxia; *cephalitis ægyptiaca*; *erythismus tropicus*; cerebral fever; ardent fever; thermic fever; sun fever; *causus ab insolatione*; *phrenitis Indica*; *typhomania*; *calenture.*

THE affections which have been described under one or other of these terms, although due to the same causes, are characterised by different symptoms, and may require different treatment. Although cases of sunstroke are recorded by ancient writers, most instances of the kind appear to have been returned in India, till a comparatively recent date, as apoplexy or remittent fever; sunstroke being reserved for those cases in which the disease occurred by direct exposure to the sun.

Among the most powerful predisposing causes are fatigue, overcrowding or bad ventilation of barracks, hospitals, or sleeping apartments, and unsuitable dress, especially unsuitable military accoutrements, and intemperance. To these have been added malaria, scorbutic taint, venereal taint, former attacks of the disease, former injuries of the head, retained excretions, defective secretions; in short, anything tending to debilitate or contaminate the system. The exciting causes are exposure to the direct rays of the sun, to great elevation of temperature either combined with dry or moist atmosphere, rarefaction of air and consequent diminished supply of oxygen—all tending to heating the body, to

evaporation of the watery particles of the blood, and to the retention of carbonaceous matter. Heat is so closely associated with other climatic conditions, such as electric, hygrometic, and barometric states of the atmosphere, telluric and other miasmata, that it is difficult to say how far heat alone is concerned in affecting those who are exposed to the combined operation of the above-mentioned agencies. Thus some authors have regarded insolation as not due to heat alone, but to other noxious agents, as the cholera pestilence, or malaria concentrated in a sufficiently potent form to destroy the vitality of the blood. But we know that the effect of heat on animals is, first to accelerate the circulation and eventually to stop the breathing and produce suffocation, probably partly from over-heating of the body, causing vaso-motor paralysis, and partly from the consequent venalised blood. And although experiments on animals must be received with caution, for it is not heat alone but solar heat which causes insolation—proved by the fact that stokers, iron-puddlers, and others who work in as great heat artificially caused do not suffer from insolation—still experiments on animals so far as they go prove the deleterious effects of heat on vitality. As we know little or nothing of the injurious effects of electric, hygrometic, and barometric conditions; as sunstroke has occurred when these conditions were very opposite; as we know little or nothing regarding telluric and other miasmata; but as we do know something of the effects of elevated temperature and rarefied air, I am content for the present to ascribe insolation to such effects. Such causes are, I believe, quite sufficient to induce insolation, even when there have been none of the predisposing causes previously mentioned in operation; but, as a rule, in epidemics of the disease one or more of the five predisposing causes first mentioned will be found to have operated.

The action of solar heat as the immediate or exciting cause may be rationally and physiologically explained. It

appears to act in two distinct and different manners. Whatever other influence may exist, it is certain that prostration of nervous power is always more or less present as the result of great heat. Some victims perish so rapidly that the result must be ascribed to syncope from nervous prostration. Great heat applied to the whole body tends to induce death by syncope, as happens after extensive superficial burns. The premonitory symptoms of insolation, as debility, weariness, vertigo, nausea, incontinence of urine, are all indicative of nervous exhaustion. But heat also acts in another and different manner. It is known that heated expanded air contains less oxygen in a given space. At 80° Fahr. a man receives 9 per cent. less oxygen than at 32° (Parkes). Smith[1] has shown that all respiratory phenomena are lessened during a hot season—the higher the temperature the less carbonic acid is exhaled—evidently a result of the smaller proportion of oxygen passing into the system. And when insolation is established the action of other organs as compensating excretors is either prevented or lessened. Perspiration is absent or partial, urine is scanty or suppressed, the bowels are often confined, while copious black stools, so often passed during convalescence, demonstrate the amount of effete material retained. In all recorded instances of insolation there has been a great, and to the Europeans an unnatural, elevation of atmospheric temperature, a consequent rarefaction of air, a diminished supply of oxygen, and interference with the compensating excreting action of various organs. Therefore, in addition to direct nervous prostration, there is the oppression resulting from venalised blood. The state of system arising from fatigue, and atmospheric impurities, as those prevailing in the air of unduly ventilated barracks, and hospitals, or jails, from the presence of alcohol in the blood, from disordered or diseased viscera, from retained excretions, will certainly add

[1] 'On the Phenomena of Respiration,' *Philosophical Transactions*, 1869.

to the venalised condition. Hence their influence as predisposing or aggravating causes. The blood, previously deprived of its watery constituents by profuse transpiration prior to the suppression of perspiration, is no longer fitted to carry on and support the functions of life, while the depressed nervous centres can no longer supply the stimulus needed. Hence the occurrence of formidable congestions in the lungs and internal organs. The disease is thus neither due altogether to nervous prostration, nor to the accumulation of effete material in the blood, but to *both* causes; both conditions arising as the results of great heat, and both reacting on each other.

The influence of *fatigue* in predisposing to insolation has been frequently painfully demonstrated. Amongst numerous other instances, the march of the 43rd Regiment in 1857 and 1858 from Bangalore to Calpee may be referred to. When 960 miles had been accomplished, the men were greatly exhausted, and numerous cases of insolation occurred.[1] On May 6, 1860, a detachment of the 4th King's Own left Ahmedabad, were out eighteen days, and lost sixteen men, the cases generally occurring when they were pitching tents after the morning's march.[2] When, during the mutinies, a detachment of the 35th Regiment lost ninety-six men from sunstroke, and retired from Arrah, the retrograde movement was caused by fatigue and sun, not by the enemy. Other evidence to the same effect may be found in Maclean's evidence before the Royal Sanitary Commission of Enquiry into the State of the Army in India.

Instances when the disease occurred epidemically from *overcrowding* in barracks or from want of ventilation are her Majesty's 3rd Dragoons at Cawnpore,[3] her Majesty's 19th Regiment at Barrackpoor,[4] the 3rd European Light Cavalry

[1] Barclay, *Mad. Med. Jour.*, 1860.
[2] Gorringe, *Trans. Bom. Med. Phy. Soc.*, 1861.
[3] Hill, *Ind. Ann. Med. Sci.*, No. 5. [4] Longmore, *Lancet*, March, 1859.

at Mean Meer; in the description of which attack Butler[1] states, those barracks most crowded, least ventilated, and worst provided, furnished the greatest number of fatal cases. At these times of epidemics among troops the officers, living in separate houses, did not suffer.

As regards the adverse influence of *unsuitable clothing* and accoutrements, Maclean[2] has collected a number of attacks in regiments marched during the heat of the day with accoutrements and dress unsuited to the climate. The influence of the old military stock which obstructed circulation through the neck, and of the belts across the chest which obstructed the action of the viscera and assisted in congesting the lungs, and of the tight-fitting coatee which acted in a similar manner, have been long since pointed out by military surgeons as some of the most potent causes of sunstroke.

The care now taken in India not to fatigue the men more than unavoidable on the line of march, the substitution of railway travelling for marching between many points, the better description of barracks now afforded, and the care taken in their ventilation, a more rational military dress and accoutrement, less intemperance, and better habits of life generally, have done much to decrease sunstroke in the Anglo-Indian army.

Insolation is most prevalent when hot weather commences suddenly after cold. Also in those calm sultry days when the sun is obscured by a film of clouds or impalpable dust,[3] or when that peculiar state of atmosphere is more than ordinarily noticeable in which the hair stands on end, and emits sparks when brushed. Although no fixed temperature can be specified as the point at which the severe type of the malady begins to show, the degree of 96° Fahr. should

[1] Butler, *Ind. An. Med. Sci.*, vol. xii.
[2] Reynolds, *System of Medicine.*
[3] Gordon, *Med. Times and Gaz.*, 1857.

cause a watch for it.[1] Difference of opinion has prevailed as to whether hot dry atmosphere or hot moist atmosphere is most conducive to the malady. Maclean observes it would appear that a hot moist condition of air is most favourable, because both evaporation and artificial cooling are interfered with. Fayrer says ardent heat alone, especially when the atmosphere is loaded with moisture so as to prevent evaporation from the person, is the real cause of the disease; but others have held the opposite view, and Day[2] stated, should any moisture occur in the atmosphere, the number and severity of attacks begin to decrease. The truth is, that both hot dry air and hot moist air are perhaps equally pernicious, although in a different manner; for while the former by exciting too great evaporation from the surface deprives the system of water, the latter by preventing evaporation adds to the internal heat. However this may be, there is no doubt that severe epidemics of insolation have occurred in both conditions of the atmosphere. It would also appear that almost any change is beneficial; the advent of a *hot* wind after a sultry calm has been known to remove an epidemic,[3] and a similar influence from moisture occurring has already been noticed.

Classes most subject.—Europeans in tropical climates are more subject to the disease than natives, although the latter do occasionally die from it. It has been asserted that the temperature of Europeans is slightly higher than that of natives; but this has not been proved in India. Livingstone, however, stated that the bulb of a thermometer under his tongue stood at 100° Fahr., while under the tongue of Africans it was 98°; but Livingstone, if I recollect right, was constantly suffering from a febrile condition. A more probable explanation may be the fact of the more abundant cutaneous excretion of carbon by the darker races, it having

[1] Bonnyman, *Edin. Med. Journ.*, May, 1864.
[2] Day, *Ind. An. Med. Sci.*, No. 13. [3] Day, *op. cit.*

been ascertained by experiment (Smith) that dark skins excrete CO_2 more abundantly than white. Also the adaptation by the dark races of their mode of living to the climate, and the alteration of their constitution and temperament during the lapse of ages to the climate (*vide* p. 23) must exert a preservative influence. However this may be, it is quite certain that a European exposed to the sun as natives often are—frequently with bare heads and bodies—would suffer from some form of sunstroke. And this, notwithstanding the fact that Europeans in good health, and well protected, have been known to stand the sun, at least temporarily, as well as natives. But they cannot stand the close atmosphere in which so many natives habitually sleep, their larger lung-capacity and their small excretion of carbon by the skin demanding probably more oxygen than the native. Next, women have been supposed less liable than men, which idea doubtless arose from a smaller number of women suffering as a sequence of less exposure of the sex. Fresh arrivals in the tropics are specially liable. In many epidemics occurring among troops the young soldiers suffered most. This, however, is explainable by the fact that fresh arrivals in the country as a rule do not take care to guard against exposure, until they learn from bitter experience the necessity of doing so. They laugh at the sun, they brave his power, and they suffer in consequence. Age indeed does not appear to exert much influence, nor does the disease attack the weakly and spare, in preference to the strong and robust. The intemperate, however, are especially liable.

Sir Ranald Martin long since recorded the capacity of resistance afforded by temperance, energy, and health. 'When in Burmah,' he wrote, 'we made a forced march of forty miles, under as powerful a sun as ever shines. The heat oppressed us almost beyond endurance, and many natives fell off their horses, vomiting, convulsed, cold, and covered with profuse clammy sweat; yet not one of us

European officers, commissioned or non-commissioned, fell sick; but we were all young, healthy, temperate, and in the high hopeful spirits of an active pursuit.' The truth of these remarks has since been sufficiently often exemplified, especially during the Indian Mutinies.

Varieties.—Morehead long ago observed, that certain terms (as previously mentioned) so generally applicable to head symptoms with coma, occurring in the hot months of the year, tend to perpetuate error, the state in question being not unfrequently produced by high temperature without direct exposure to the sun. The varieties of insolation which may be practically derived from the etiology, patho logy, and symptoms are: 1. Thermal ephemeral fever; 2 Ardent fever; 3. Heat syncope; 4. Heat apoplexy; 5. Heat asphyxia; 6. Spinal congestion.

1. Thermal Ephemeral Fever commences with heat of skin, and slightly quickened pulse after exposure to the sun, and may disappear in a few hours under the influence of a cold or tepid bath, and a night's rest. Probably, most people who have been exposed to the sun during the day experience more or less of this febrile excitation, and often take no notice of it. I know it has frequently occurred to myself. Sometimes it may cause a restless night, and require a morning aperient. Or it may continue with languor, weakness, loss of appetite, pain in the back and limbs, quick pulse and heat of skin, presenting indeed exactly the same symptoms as ephemeral fever arising from cold and wet excepting the prior chilly stage. It seldom lasts more than twenty-four hours, and then declines or developes into *ardent* fever. But during these twenty-four hours it is possible that it may be mistaken for the initiatory stage of some other disease. It is distinguished from chicken-pox by the absence of eruption; from scarlatina by the absence of sore-throat; from measles by the absence of cold in the head and cough; from small-pox by the absence of severe

vomiting, and pain in the loins and eruption; from typhoid by the sudden rise of temperature. From the commencement of remittent fever it is not easily distinguished, though thirty-six hours' observation will usually suffice to mark the difference by the subsidence of fever, or by the incomplete cessation of symptoms which marks the progress of remittent. These observations, with the fact of prior exposure to the sun, will always be sufficient to establish a diagnosis. But thermal ephemeral fever may run on into ardent thermal fever, as described below; and between the slightest degree of the one and the highest degree of the other there may be every shade.

Premonitory symptoms of sunstroke.—Frequently previous to an attack of insolation of any description except the trivial thermal ephemeral fever, and more rarely previous to an attack of ardent fever, the person affected becomes irritable, restless, and complains of headache. He feels dull and listless, and unequal to exertion. The appetite fails, there may be nausea, and the bowels are constipated. After a period of profuse perspiration, an absence of perspiration may be noticed, the skin being unusually hot and dry. There may be slight sensations of giddiness. More frequent desire to make water than usual may be noticed, which is due to altered character, rather than to excess of secretion, as little fluid is passed. Such premonitory symptoms may prevail for several hours or days previous to the developed attack, or they may not occur at all; or occurring, may pass away. Recognition of these early symptoms is of the utmost importance.

2. Ardent Thermal Fever.—As just stated, it may develope from the minor form, or after premonitory symptoms, or it may originate in full violence when the attack is sudden with sometimes premonitory chills, and often occurring in the night after a day of exposure to the sun. The descrip-

tion which Dr. Arnott[1] left of this fever, cannot be improved upon: 'Pungent heat of skin, great thirst, parched red and dry tongue, quick, full and strong pulse, racking pains in different parts of the body, acute headache with flushed countenance, throbbing of the temples, restlessness, nausea, and vomiting of bilious matters.' The duration is about sixty hours. Sooner or later collapse comes on, with more or less of those signs to which the term typhoid may be applied. While many of the symptoms of this fever are those of remittent or exaggerated ague, it is distinguished by its greater intensity at the commencement and higher temperature, and by the absence of remissions other than the slight daily ones common in every kind of illness, and by the season of the year; ardent fever occurring in the hottest weather, remittent in the autumnal season.

3. Heat Syncope.—Either after the foregoing premonitory symptoms, or without such deviations from health having been observed, heat syncope commences with faintness, sickness, and giddiness, confusion of ideas, confusion of vision, perhaps loquacity and hysterical laughing or crying, cold extremities, frequent desire to micturate, and sometimes drowsiness. The face is pale, the surface cold and often bathed in perspiration, although this depends in a great measure on the dryness or moistness of the atmosphere. The respiration is of a sighing or gasping character, the heart's action and pulse are weak, sometimes intermittent; the pupils are contracted, and there may be more or less decided insensibility. In this condition there is no stertor, and the patient dies, sometimes in a hour or less, from syncope. But if the case is prolonged, the pupils become dilated, and there may be sonorous mucus râles, and the person dies asphyxiated, or he may pass into a state of coma and die from heat apoplexy. Heat syncope is more likely to occur to persons having naturally weak hearts.

[1] *Med. Hist of the Bombay Fusiliers*, 1860.

4. Heat Apoplexy; True Sunstroke, or Coup-de-soleil.—Heat apoplexy may be preceded for a variable time by the *premonitory symptoms* above detailed; or *secondly*, it may commence as *syncope*, which condition after a few hours or even minutes passes into another state, characterised by flushing of the face, heat of body and head, bloodshot eyes, strong quick pulse, stertorous breathing, with insensibility and sometimes convulsions. Or *thirdly*, heat apoplexy may occur suddenly without either premonitory symptoms or preceding syncope. In such cases the person falls down suddenly as if struck by apoplexy, and the symptoms are as above described, commencing with flushing of the face. Sometimes there may be convulsions, but in the majority of cases the person does not move again.

5. Heat Asphyxia may or may not be preceded by premonitory symptoms. It usually occurs suddenly in the night, and men have frequently been found dead in the morning from this form of heat affection. It is characterised by gasping, noisy, laboured, irregular breathing, quick small pulse and heat of skin, with insensibility more or less complete. Sometimes convulsions occur.

6. Spinal Congestion.—The part which congestion of the spine plays in the different modifications of insolation has received very little attention from Indian authors. Yet there can be no doubt but that this organ is often implicated to a very large extent. Perhaps in a minor degree to the brain (owing to the mass of bone and muscle which nature has placed over the part) the spinal marrow must certainly be remarkably sensitive to external influences, and especially to the action of solar heat. The integument of the whole back is abundantly supplied with nerves, which taking their origin from the spine, convey impressions to that organ from a very extensive surface. If the back be exposed for a short period to an ordinary fire in such proximity that the thermometer would rise to 100°, unpleasant feelings of faintness or

sickness are experienced by most persons. Several of the symptoms noticed in one or other form of sunstroke, or of the premonitory symptoms, may be correctly and directly referred to spinal irritation. Thus some amount of the difficulty of breathing observed during heat asphyxia is probably attributable to more or less paralysis of the intercostal nerves, hindering a proper expansion of the parietes of the chest. Similarly, the irritability of the bladder, hysterical nervous spasmodic movements, and convulsive attacks, also demonstrate implication of the spinal column. Mon. Duclaux gives an account of various phenomena produced by excessive heat in reapers, whose position exposed their backs to the sun. These persons were suddenly attacked by headache, muscæ volitantes, failure of strength, particularly in the lower limbs, uneven gait, giddiness and syncope.

During convalescence from all forms of insolation, large quantities of blackened fæces are often passed.

In the description of the symptoms it may be observed that the principal differences in the forms are, *first*, the amount of fever; *secondly*, in the appearance of the face; *thirdly*, in the pulse; *fourthly*, in the respiration. In ardent fever, the fever is the principal feature. In heat syncope the countenance is pale, and the surface, particularly of the hands and feet, feels cold; in heat apoplexy the face is flushed, and the surface warm or hot. In heat syncope, the pulse is weak and often intermittent, and there is sighing, irregular, gasping respiration. In heat apoplexy the pulse is strong and quick, and the breathing is stertorous. In heat asphyxia there is hot skin, quick pulse, and noisy laboured breathing; but not stertorous breathing from the first. In heat syncope and heat apoplexy the primary condition appears analogous to *shock*. In heat asphyxia and ardent fever, shock is not so prominently declared.

Post-mortem appearances vary, according to the form

of the disease. In death from ardent thermal fever, serous effusions in the ventricles and between the membranes of the brain have been noted, with turgescence of the vessels. The pulmonary system is also congested. In death from heat syncope, no remarkable morbid change may be noted, but sometimes the lungs and brain are congested. After death from heat apoplexy, the brain and membranes are congested, especially the large vessels and sinuses; there is a large amount of fluid in the ventricles, and many vascular points on the cut surface of the brain. Arndt, however, believes that those writers who have described hyperæmia of the brain as occurring in insolation have been misled by the fact that on cutting the organ the blood escapes from the large vessels, and then flows over the surface of the brain; a conclusion not very complimentary to the numerous pathologists who have described a considerable amount of brain-congestion in at least one form of insolation, and which undoubtedly exists. After death from heat asphyxia, congestion of the lungs, bronchi filled with frothy mucus, and distension of the right side of the heart, with coronary arteries much distended, are the principal morbid appearances. Hæmorrhagic spots have been found in the stomach, bowels, and bladder in nearly all cases, the heat of the body after death is inordinately high, and *rigor mortis* is rapid. Sometimes there are patches of ecchymosis, or of erysipelatous blush on the skin. The blood-globules are sometimes found crenated and the blood fluid.

When the medulla is more affected, accumulation of blood takes place in the right heart and lung, with, secondarily, as a consequence, a want of that fluid duly arterialised in the brain. There is death from the first, without declared shock, commencing at the lungs, or the asphyxiated condition taking place gradually, while in the other varieties death results from failure of the action of the heart, sometimes depending on tetanic spasm, or from syncope, or from

coma. It is true mixed forms of insolation are frequently met with, and if the causes of the malady are considered it may be understood how this should be the case. High temperature with its direct sedative effects, the quick evaporation from and inspissation of the blood, the rarefaction of the air and consequent diminished supply of oxygen at each inspiration, and the resulting venalisation of the blood, together with debility, fatigue, depression, excitement, are certainly likely to predispose and excite both nervous and circulatory disturbance.

Mortality.—This is high. Death may take place at all periods, from ten minutes to eighteen hours after seizure. The number of deaths in proportion to attacks has been variously estimated. Bryden states that from 1857 to 1867 the mortality was 51·37 per cent. Other authorities have estimated it at 43 per cent.

Treatment.—It was formerly generally maintained that the primary lesion in insolation is active congestion of the brain, but this view has been vigorously attacked in recent years, especially by Arndt, whose remarks have already been referred to. It is now admitted that in all forms of the disease, although less so in ardent fever, there is exhaustion from heat, and this fact should be prominently borne in mind. For a minor degree of sun fever, or for irritability after exposure, a cold or tepid bath according to habit, rest and quiet under a punkah, and if the bowels were previously confined an aperient dose, are desirable. In the treatment of ardent thermal fever we must rely on cold affusion, diaphoretics, diuretics, and aperients, with perfect rest and quiet, followed if necessary by wine, brandy, or ammonia; particular congestions or inflammations arising being treated generally or locally as required. When heat syncope is present, or when the patient is faint, sick, and giddy, shivering and cold, it will be generally advisable to lay him on the back in the shade, to rub the limbs, to

loosen the clothing, to give a stimulant in the shape of wine or brandy and water, and to apply warmth to the extremities. But the case must be treated with caution on account of the tendency of the condition to run on to that described as heat apoplexy.

When flushed face, heat of skin, bloodshot eyes, quick strong pulse, or stertorous breathing are observed from the first, cold water should be poured on the head, punkahs should be used to cool the surrounding atmosphere, and if the patient can swallow, a quick purgative, or if he cannot swallow, croton oil placed on the tongue should be used. If available, eight or ten grains of quinine should be added to the purgative draught. An assafœtida injection should be given cold, or, if not at hand, a cold water injection. The extremities should be rubbed, mustard poultices or turpentine stupes should be applied to the nape of the neck, and if coma ensue the injection should be repeated, cupping should be performed on the back of the neck, or twenty leeches may be applied to the margin of the hair. The patient may also be wrapped in a wet sheet. But great care should be taken not to prolong cold applications too far, as danger may result from depression of temperature below the normal standard of blood heat. If the skin refuse to act and maintains its high temperature, Maclean recommends a trial of Warburgh's tincture, which is the most powerful sudorific with which he is acquainted.

When heat asphyxia is present, shade, position, cold affusion, cool atmosphere, friction of extremities, quick purgation, turpentine or mustard to the nape of the neck and to the chest, and the application of the wet sheet are the principal means of relief. In the mixed form of the disease, when both brain and lungs appear implicated in the same degree, mustard or turpentine should be applied both to the back of the neck and the chest.

In all varieties of sunstroke the patient should be

encouraged to drink plentifully of cold water (or if available a little chlorate of potash should be mixed with it), to supply the place of the fluid constantly passing by perspiration or evaporation from the skin. It must be recollected that the patient is not free from danger until the skin is naturally cool and moist.

In this disease, as in many others, the propriety or otherwise of bleeding has been much discussed. During the Indian Mutinies, when so much experience was gained of the malady, Longmore[1] and Pirrie[2] found bleeding of no use, while Butler[3] feels assured some of his cases would have died without it; and so adverse opinions might be quoted in great numbers. The fact is, it is quite impossible to lay down stringent rules for the treatment of the different forms of insolation. The constitutions of men are diverse, and they suffer under varying circumstances of fatigue, excitement, and depression; epidemics differ in degree and characteristics; and such facts may perhaps account for remedies found effectual at one time and place being the reverse at another. In this disease, as in most others, the treatment should be deduced from a rational and judicious consideration of the symptoms, circumstances, and conditions presenting. The extreme exhaustion attending, and perhaps causing insolation is admitted, and, as before stated, this should never be absent from the mind of the surgeon. But, on the other hand, this knowledge should not cause him to flinch from the use of depletory measures, if the condition of the patient demands them. Recent writers go too far when they assert that abstraction of blood in any manner is never necessary, and that 'the congested livid surface, coma, and stertor which formerly suggested bleeding, do so no longer.' Surely the quick and effectual relief of congestion by abstraction of blood is demanded in some of these cases of sunstroke, to

[1] Longmore, *Lancet*, 1859. [2] Pirrie, *ibid.*, 1859.
[3] Butler, *Ind. An. Med. Sci.*, vol. xii.

avert suffocation. Loss of blood may be regarded as an eventual evil, but in treating this disease it is sometimes necessary to choose the least of two evils. Fayrer truly observes, 'It is possible there may be more danger to life from a labouring and distended heart and embarrassed lungs, than from the loss of a few ounces of blood which, if it tided the patient over that danger, as I believe it sometimes might do, would be the lesser evil.' Each case must be treated on its own merits. In plethoric persons suffering from heat apoplexy or asphyxia, from stertor, coma, and engorged lungs and heart, I recommend, as the result of experience, the abstraction of a small quantity of blood. This measure does not prevent the use of stimulants and cold affusions, neither is the combination unscientific.

Other suggestions which have been made for the treatment of insolation are emetics of ipecac,[1] which may be useful if the disease occurs after a meal and there are attempts to vomit; and the subcutaneous injection of quinine if the patient cannot swallow.[2] The power of quinine in modifying the dangerously high temperature which attends sunstroke seems well established, and the patient may be rapidly brought under its influence by the hypodermic method,[3] so the measure, as it can do no harm, should not be neglected. For convulsions chloroform has been used, but is not recommended, as it must increase internal congestions.

Engaging the patient's attention in loud talking is another suggestion which has been practised, when rest and tranquillity are more required. The ready method for restoring the drowned has also been used, but without sufficient success to render it a part of practice.

It is highly desirable to ascertain if the patient has

[1] Gorringe, *An. Rep. of Fourth Ret.*, 1861.
[2] Waller, *Ind. Med. Gaz.*, July, 1869.
[3] Maclean, *Army San. Rep.*, 1870.

previously suffered from venereal. If so, iodide of potassium in large doses should be given as soon as possible, as syphilis, already mentioned as a predisposing cause, sometimes produces a condition of the nervous tracts which may perhaps predispose to insolation, and probably aggravates its after-effects. When amendment is once established the sooner the patient can be induced to take nourishment, such as good broths, &c., the greater the chance of recovery.

Although recovery is often rapid and complete, more commonly fever and oppressed breathing prevail more or less for some days, from which the patient gradually recovers. Sometimes during convalescence there are a succession of dark bilious stools. Sunstroke is, however, frequently followed by periodical headache or neuralgia, by dysentery, or by tendency to fever not previously developed. Paralysis may also occur as a sequel, and the affection lays the foundation of a number of cases of insanity, probably with softening of the brain. Even should recovery seem complete, it often happens that the individual is unable afterwards to bear exposure to the sun, and thus becomes unfitted for active life in a tropical country, and probably eventually suffers from *ramollissement.* These sequelæ of insolation may be attributed to the injury the brain suffers during the primary attack, probably from loss of nutrition in the one case (syncope) and from congestion in the other (apoplexy), by which it may be weakened for life. But this direct sunstroke by day, and deferred sunstroke by night, though the most appalling, are not the most extensive effects of the sun. There are thousands who escape these attacks, but whose constitution is nevertheless ruined by sun-heat (*vide* anæmia, p. 18), who are rendered by it liable to every form of tropical malady, who are unable to survive even slight wounds or operations, not so much from effects on the brain as from exhaustion of the skin, for as Julius Jeffreys long since

observed as a pathological axiom, 'the skin's debility is malaria's opportunity.'

Means of Prevention.

Avoidance of exposure to direct solar heat; wearing proper head-dresses, or such as fit tolerably closely to the scalp, by which the perspiration is confined and the head kept moist. The widely ventilated hats so vaunted by their contrivers do not fulfil their purport, especially in the districts when and where hot winds blow; the wide apertures round the circumference allow the hot winds to pass freely over the cranial surface, keep it constantly dry, and thus predispose to attacks of the malady they were designed to prevent. Wetting the head-dress with water when obliged to go out in the sun, or wearing a plaintain leaf inside; wearing a shade or pad for the upper part of the spine, which may be attached to the coat; using a white covering which, even in the shade, reduces the temperature 2°, and in the sun from 130° to 111°.[1] Wearing loose clothing round the neck and chest; the artificial cooling of dwellings by punkahs and tatties; avoiding overcrowding, especially of sleeping rooms; sleeping in the open air or in the verandah in the hot season; abstinence from, or at least a very moderate use of, spirits.

Among troops, in addition to the above general precautions, confinement to the barracks during the heat of the day has been advised, which, however, is a questionable measure.

Serving porter in barracks, instead of at the canteen, in the middle of the day, diminishing the number of European sentries; parades at 4½ A.M. and 6 P.M. Exempting officers from day visits to barracks; pitching tents for sleeping in to relieve the barracks; placing vessels filled with cold water at hand for douching, and instructing non-commissioned officers in their use: marching troops in very open order by which ventilation in the ranks is secured, a source of much comfort as all will allow who have ever made a hot and dusty Indian march: the provision of privy tents for the men on the march.

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[1] Sykes, *Jour. Stat. Soc.*, vol. xv.

CHAPTER XXI.

JAUNDICE.

JAUNDICE, although usually described as a disease *per se*, should rather be regarded as a symptom in the same light as albuminuria or oxaluria are viewed. Jaundice being associated with a variety of morbid states, as diseases of the liver, fevers, poisons, &c., cannot be regarded as dependent on hepatic derangement alone, although it is with affections of the liver and its appendages that it is most frequently connected. It is yet a debatable question how bile is formed, although analogous reasoning, as in the case of the kidneys for instance, would lead to the belief that bile is formed in the liver by the action of that organ on the blood. But some physiologists are of opinion that some of the elements of bile are formed in the blood, and others in the liver, the function of the latter organ being principally selective. I am not, however, inclined to these views. I think it is scarcely possible to formulate a theory of jaundice other than that bile-pigment is first secreted and then reabsorbed, and I thus am not inclined to draw the distinction which has been made of *hæmatogenous* and *hepatogenous* jaundice.

The most simple division of jaundice is perhaps that adopted by Murchison. 1. Cases in which there is a mechanical impediment to the flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages and thence absorbed into the blood. 2. Cases in which there is no impediment to the flow of bile

from the liver into the bowel. Those physiologists who regard the liver as simply a secreting organ hold these latter cases of jaundice to be due to suspended secretion from functional disorder of the liver. It has not, however, been satisfactorily shown that bile-pigment exists ready formed in the blood, either general or portal, of persons who have not jaundice. The secreting tissue of the liver may be so destroyed that little or no bile can be secreted, but no jaundice results. After extirpation of the liver in the lower animals bile does not accumulate in the blood as urea accumulates after extirpation of the kidneys. The most probable solution of the difficulty appears to depend on the fact that a large proportion of the bile which is secreted is again absorbed by the biliary passages or the mucous membrane of the bowels. Under normal conditions the whole of the bile that is absorbed is at once transformed, so that neither bile-acids nor bile-pigment can be discovered in the blood or in the urine, and there is no jaundice. But in certain morbid states the absorbed bile does not undergo normal metamorphosis, but circulates in the blood and stains the skin. According to this view the only pathological difference between jaundice from obstruction and jaundice without obstruction is that in the former case little or none of the bile secreted by the liver can escape with the fæces, so that all, or nearly so, is re-absorbed, the quantity being too great to undergo normal metamorphosis; while in the latter case bile passes into and is discharged from the bowel as usual, but that which is absorbed, which in quantity may not exceed what is absorbed in health, remains unchanged in the blood. The morbid states which appear to conduce to this result are certain poisoned conditions of the blood, such as those of yellow fever, relapsing fever, remittent, typhus, typhoid, snake poison, &c.; nervous influences—as sudden fright, rage, protracted anxiety, and injuries of the brain; a deficient supply of oxygen, as happens in certain cases of pneumonia, or in

persons living in confined and crowded dwellings; and excessive secretion of bile, especially when accompanied by constipation.

Under the views of the pathology of jaundice as above, the causes may be stated under the following headings:— 1. Jaundice from mechanical obstruction of the bile-duct includes: A. Obstruction by foreign bodies within the bile-duct. B. Obstruction by inflammatory tumefaction of the duodenum or bile-duct. C. Obstruction by stricture or obliteration of the duct. D. Obstruction by tumours closing the orifice of the duct. 2. Jaundice, independent of mechanical obstruction of the duct, includes: A. Poisons in the blood. B. Impaired or deranged innervation. C. Deficient oxygenation of the blood; all interfering with the normal metamorphosis of bile. D. Excessive secretion of bile, more of which is absorbed than can undergo the normal metamorphosis. E. Undue absorption of bile from habitual constipation. Under No. 1 and its divisions some thirty special causes may be enumerated; under No. 2 and its divisions a still larger number.

Jaundice, as seen in India, may be simply divided into temporary and permanent, the former met with in connection with nervous influences and fevers, or from catarrh of the ducts or inspissated bile, from calculi, entozoa, impacted fæces, or perhaps filaria—the latter met with in connection with chronic affections, especially of the liver.

The ordinary *symptoms* of jaundice are as simple as the pathology is obscure. The skin becomes more or less uddenly greenish or yellow, which has led to the malady being spoken of as the 'green' or 'yellow' jaundice, as if there were two distinct kinds; in some cases the colour is so deep as to have given rise to the term 'black' jaundice. The difference of colour, however, merely depends on the amount of bile contained in the blood, and when jaundice does not arise from obstruction it is rarely very deep. The whites of

the eyes assume a greenish or yellow tint, and this is of course more apparent in the native than the cutaneous discoloration. . Vision is often affected, everything appearing yellow (xanthopsy). The bowels are constipated; but in some cases, in the absence of bile, the fæces, becoming putrid and irritating, cause diarrhœa. The motions are white or clay-coloured. The urine is yellow and usually presents colour before the skin, and in slight cases the jaundice may be limited to the skin and conjunctivæ. The skin usually itches generally or locally, and the pruritus is worse at night. Other secretions, as the milk, saliva, tears, or the serum from a blister may contain bile. When pneumonia exists with jaundice there is often bile in the sputa. There is a bitter taste in the mouth, especially in the morning, due to taurocholic acid, as bile-pigment is tasteless. There is also coated tongue, nausea, and flatulence. The person's temper is irritable, and he is depressed and melancholy. The temperature is not increased unless the jaundice is associated with fever, and the pulse is slow and often irregular. The digestion of fat is interfered with by the absence of bile, and fat accumulated disappears and emaciation takes place. Occasional symptoms are its sudden appearance with rigors, vomiting, and hepatic congestion; the occurrence of purpura and hæmorrhages, owing to the blood becoming impoverished from less number of red corpuscles and diminution of fibrine, or from scorbutic taint; nettle-rash; jaundice limited to one-half the body, or presenting in yellow spots or streaks on the flexures of the limbs. Cerebral or cardiac symptoms may also occur, such as staggering, drowsiness, partial spasms, convulsions, delirium, coma. In such cases the typhoid condition may present. There is some reason to believe that cerebral symptoms are caused not by the bile itself but by the injection into the tissues of decomposing mucus contained in the bile. But a tendency to head symptoms suggests acute atrophy of the liver, in which the morbid

change is a parenchymatous degeneration, consisting of the filling of the hepatic cells with albuminoid granules and oily particles. The functions of the liver are thus further impaired. Albuminous matters are not disintegrated into urea and uric acid, leucin and tyrosin take the place of urea in the urine, and cerebral symptoms are prominent features.

The occurrence of purpura or hæmorrhages during jaundice implies an anæmic state, probably depending on scorbutic taint.

When jaundice is accompanied by rigors the presence of a gall-stone may be surmised, the symptoms of which, unless explained by the jaundice, are sometimes difficult to distinguish from the passage of renal calculus. There is excruciating pain immediately to the right of the pit of the stomach, shooting to the back, with vomiting of sour bile. From this pain there are intervals of comparative ease, and pressure will relieve it to some extent, the person throwing himself about the bed, or pressing his thighs on the abdomen to obtain relief. This distinguishes the malady from inflammation when pressure and change of posture are painful. The absence of retracted testicle, of pain shooting down the inside of the thigh, of numbness of the leg, of pain in the loins, and of frequent desire to make water, are the chief features distinguishing gall-stone without accompanying jaundice from the passage of gravel or renal calculi. The previous history should also be taken into consideration, as both hepatic and renal calculi are apt to recur.

The cause of the symptoms enumerated as those of jaundice is the presence of bile in the blood. The skin and kidneys being the great eliminatory functionaries of foreign bodies, the bile, or at least the bile-pigment, flies to them. Urine containing bile treated with nitric acid on a white plate gives a play of various colours; treated with hydrochloric acid it becomes olive green; two or three drops of strong iodine tincture added to an inch of urine in a test-

tube affords a bright emerald green at the point of contact. Bile-acids are sometimes found and sometimes not, which Harley explains, because in jaundice from suppression the liver does not secrete bile, consequently no bile-acids being formed none can enter the circulation; but in jaundice from obstruction bile is secreted and reabsorbed into the blood, a portion of the bile-acids being then eliminated by the kidneys. Harley considers the question whether jaundice is or is not due to obstruction can be determined by the presence or absence of bile-acids in the urine. Adopting the view that bile-acids are formed by the liver while bile-pigment is preformed in the blood, he contends that in jaundice from suppression, or independent of obstruction, the liver does not secrete bile, and consequently no bile-acids being formed none can enter the circulation or be detected in the urine; whereas in jaundice from obstruction bile is secreted and reabsorbed into the blood, and a portion of the bile-acids not transformed in the circulation appear in the urine. But in addition to the strong improbability of any form of jaundice being due to suppressed secretion of bile, Murchison stated bile-acids have been found when there is no obstruction, and in many cases of mechanical jaundice they are absent. A more reliable indication is furnished by the stools; when there is no obstruction the stools almost always contain bile: when the duct is obstructed they are white.

Slight jaundice may require diagnosis from chlorosis, from the anæmic aspect of visceral or malarious disease, from the bronzing of Addison's disease. The test of nitric acid applied to the urine, and the appearance of the stools, are conclusive.

The jaundiced appearance is sometimes produced by malingerers by eating turmeric, which, taken in large quantities, gives a sallow expression to the countenance and colours the urine. The yellow particles may, however, be detected in the fæces. Picrate of potash and santonine also produce a yellow reddish cast in the urine, the latter agent also producing yellow vision. But the stools are the crucial test, as

they are not affected by either of the agents named. It should, however, be recollected that when jaundice is really present the stools may present a dark appearance, from either blood or from sulphuret of iron, derived from medicines taken.

ICTERUS CATARRHALIS, attended or not with hepatic congestion, is perhaps the most frequent form of the disease seen in India, not taking into consideration jaundice occurring during fevers and from chronic liver-disease. This arises from the stagnation of viscid bile, and answers to the icterus spasmodicus of Cullen. Inspissated normal bile is sometimes so dense that it has been mistaken for gall-stones, which are, however, composed not of bile but of cholesterin, being unyielding, not brittle, like inspissated bile, and often light in colour like a pearl, while thickened bile is always brown. In *treating* any variety of jaundice the cause must first be sought. If there is no fever, no chronic liver-disease, no gall-stone, no impacted fæces in the colon, the cause of the malady will most probably be inspissated bile in the ductus communis choledochus, which prevents flow into the duodenum. In such cases an occasional mercurial, followed by saline cathartics, alkalies and taraxacum, with the nitro-muriatic bath are desirable. The propriety of giving mercury in such cases has, however, been debated chiefly on the grounds that mercury does not act on the liver in dogs, and that the dark stools following mercury result from a sulphuret of the mineral formed in the intestines. This I do not believe, for I maintain that mercury produces a flow of bile, whether from direct action on the liver or pancreas, or, as has been argued, by stimulating the duodenum and by reflex action on the gall-bladder, I do not know. The above treatment, however, should not be continued too long. If no amelioration in a few days, it may be changed for nitro-muriatic acid, in combination with podophyllin. Or euonymin or iridin may be tried.

In treating the calculous form of the disease, chloral or

opium and the warm bath are the principal remedies. If the icterus arises during the progress of other diseases, the latter, rather than the jaundice, should be treated by the appropriate remedies. The mineral acids are perhaps the best remedies in cases of jaundice depending on fevers or other forms of blood-poisoning.

CHAPTER XXII.

LEPROSY.

CONSIDERABLE confusion has existed as to what leprosy really is. The disease has been known by many names, either derived from the symptoms or the results. Among these are elephantiasis anæsthetica, elephantiasis Græcorum, leontina, mutilans, nodosa, satyria, joint evil, myckle evil (old English), or the great disease. Leucoderma has also been described as a form of leprosy, which appears to have arisen from the expression used in the Bible, 'as white as a leper;' persons therefore affected with an absence of pigment occurring in patches over the surface of the body have been supposed to be suffering from leprosy. But when leprosy is spoken of in Scripture it is spoken of indefinitely, at one time as a minor or probably any skin disease, in other places as a serious disorder: an error which has been perpetuated. The term elephantiasis appears to have arisen from the idea that leprosy was as much greater than other diseases as the elephant is greater than other animals. Paulus Œgineta[1] described leprosy under the term elephantiasis, and this error has been perpetuated. The term leontina arose from the supposed resemblance of the eyebrows to those of a lion.

Dr. Adams in his commentary says the Greek translators of Avicenna rendered the Arabic word *jugum* by lepra, but they also called the Barbados leg elephantia from its resemblance to the leg of an elephant, and the Greeks, who had been accustomed to designate leprosy as elephantiasis, mixed

[1] *Sydenham Society's Translations*, Book iv.

up the idea of elephantia with elephantiasis, and thus the Barbados leg came to be considered as a species of leprosy. To make matters worse, of late years lepra, a skin disease classed by Willan under squamæ, has been described as leprosy, with which it has no relation. This application of the term lepra to leprosy may probably have occurred from the fact of the minor form of leprosy, which most resembles lepra (although, indeed, there is little similarity), having been in former times, as it is now, the most common.

We have additional evidence to that of Scripture that true leprosy has been known from the earliest times, as it is mentioned by Aræteus and other early authors; and it is regarded as more than probable that the 'baras' of the Arabs and the 'leuka' of the Greeks was leprosy.

In Europe there appears to have been a great increase of leprosy after the Crusades, thought to be due to contact with Oriental nations. In France, in 1226, Louis VII. left legacies to 2,000 leper-houses, and old records show that leper-houses were common in France, Germany, and England. At present in its endemic distribution, the malady corresponds as regards intensity with the belt of maximum heat of the globe. It chiefly affects the Hindoos, Mahomedans, and Chinese living within or on the borders of the tropics. It is also well known in the islands of the Indian Ocean, Africa, Madagascar, in the West Indies, and in South America. It is met with in a less inveterate form in Spain, and on the Mediterranean coasts, and prevails in Greenland, Norway, Iceland, Lapland, and Northern Europe generally. Thus at the present time, although leprosy is perhaps more destructive and prevalent in India than elsewhere, it still, as Carter observes, 'pertains not to country or clime.' It may, however, be doubted if the leprosy observed in cold climates is the same inveterate disease we observe in the East. Hobson[1] states that the Chinese consider a visit to the north as a certain

[1] 'On the Leprosy of the Chinese,' *Med. Times*, 1870.

cure for leprosy. As regards Western India the disease prevails only to a comparatively slight extent in Rajpootana, Sind, and Kánara, to a greater extent in Gujerát and the Southern Marátha country, and is common in the Deccan and the coast districts of Thána and Kolába, reaching a maximum in Khándesh, where there are 2,186 lepers, forming 177 per cent. of its population. In the Presidency of Bombay in 1884 (exclusive of the city of that name) there were 9,483 lepers of the worst type.

Age.—Although leprosy may occur in childhood, it is most prevalent during the more vigorous period of life. According to the best Indian authorities, 16 per cent. date their malady from childhood; but children have not been known to be born lepers, and many children of leprous parents look quite healthy. Leprosy is seldom seen in very old people, as lepers die before attaining to old age.

Sex.—According to Carter's inquiries, more men are affected with leprosy than women. It would also appear that females are most subject to a mild variety of the disease.

Race.—The prevalence of leprosy among Eastern races has already been noticed. But although leprosy is so common in India among the natives, it is, even in *ratio* to numbers, comparatively rare among Europeans. In Bombay, however, there are generally one or two European, and many half-caste lepers to be found; and it is stated that some years ago there were three European lepers in Madras 'having every comfort money could procure.' In the Mofussil I do not recollect seeing a European leper. But the immunity, if any, of Europeans ceases to appear as we leave the tropics. Probably the immunity of Europeans from leprosy in India may be explained in the same way that their comparative immunity from elephantiasis is explained (*vide* p. 236). That there is not any special immunity even of Englishmen is certain, for some years back cases were treated by Syme and Simpson at Edinburgh, by Addison at Guy's,

and at later periods by other physicians, the patients never having been out of England.

Causes.—Leprosy has been supposed to be caused by a want of salt and a purely vegetable diet, but there is no reliable evidence in support of these ideas. I have seen leprosy throughout the semi-desert districts of Western India, where the principal product of the country is salt, and even on the banks of the Great Sambhur salt lake. As regards vegetable diet, the fact of the disease occurring among the meat-eating populations shows that the malady does not originate from vegetable diet. Doubtless a want of salt and a purely vegetable diet will tend to induce an anæmic condition of system, and so may predispose to this disease as to various others, but there is no reason whatever to believe that it predisposes to leprosy especially.

A too exclusive fish diet has also been thought to be a cause, especially as the disease prevails to a considerable extent among the fish-eating populations of the Indian coasts. But the disease also prevails to almost the same extent in the interior, many miles from the coast, where the people never see sea-fish and very rarely fresh-water fish, where, indeed, fish forms an infinitesimal part of the dietary. The Banians, who never eat fish, are not exempt. Diseased grain or new rice have been mentioned as causes, but without sufficient or indeed any reliable evidence. The kind of food used does not appear to influence the disease in India, and the same may be said of occupation and caste. What Carter states of the disease in Kattywar is applicable to the whole of India, 'It attacks the poor, and spares not the well-to-do.'

The question of the *hereditary nature* of leprosy has been the subject of much difference of opinion. The writings of Danielsen and Boeck, and the reports of Virchow and of the English College of Physicians, also of Italian observers, seemed to show that leprosy was hereditary, and Khory says

it is undoubtedly transmissible from parent to offspring. But since the date of these reports the conclusion has been disputed. Hobson states in the majority of cases evidence of heredity is absent; and Hansen, in Norway, has attacked the view of its hereditariness. He notes that in a district where leprosy is of recent origin the indications of heredity are slight or absent, and that it is not until the disease has become endemic that the supposed proof is forthcoming. Dr. White, of Harvard University, has also recently concluded that heredity is not an all-important factor in the propagation of leprosy. In India Morehead may be quoted as against the hereditariness of the disease. Lewis and Cunningham also came to the conclusion that heredity could not be a great factor in the increase of leprosy in a district, inasmuch as lepers have comparatively small families who suffer a high rate of mortality, and therefore the survivors are only just numerous enough to replace their defunct progenitors. Carter[1] states the hereditary taint is comparatively rare in Kattywar, that heredity as the exclusive agent in the propagation of this disease does not seem entitled to the position once allotted to it, and even considers heredity to be doubtful, for although 30 per cent. have a direct or collateral taint they have also opportunities of contracting it by contagion. Carter also mentions the fact that all lepers have not leper children. It is indeed a matter of common experience that only one or two of the children of a leper may be attacked, and these not in any regular order. Then again, children are never born with the evidences of leprosy upon them, although cases have occurred of the development of leprosy during infancy.

As with the question of heredity so with that of *contagion* —difference of opinion prevails. Popular belief has pointed, at all times and in all countries, to the propagation of leprosy by contagion, and all the arrangements for leprosy in the

[1] *Modern Indian Leprosy*, p. 73.

Middle Ages were based on this assumption. But the reports of Danielsen, Boeck, Virchow, and of the English College of Physicians, previously referred to, tend to prove the heredity and not the contagiousness of the malady. Chevers is unacquainted with any fact in support of contagion. Rustomjee Khory observes, 'It is not contagious; the attendants on the sick do not contract it, nor does sexual cohabitation impart it to the healthy.' But Hansen, in Norway, has produced strong evidence in favour of contagion. It has also been noted that the disease only appeared among the Sandwich Islanders after Chinese emigration, and at Surinam, and in America after the importation of negroes. Munro, in his work on leprosy, supports the contagiousness of the affection. Carter, giving more weight to positive than to negative statements, is of opinion that leprosy is contagious, meaning the direct or indirect conveyance of the disease outside the body from person to person. Liveing, while not regarding it contagious in the ordinary sense, thinks it might be imparted by imbibition of the excretæ of lepers, as cholera and enteric are supposed to be communicated. In support of the contagious nature of the disease Dr. Atkinson's case may be quoted of undoubted tuberculated leprosy in a married woman of German parentage, and who had never left the State of Maryland. Her children and husband were healthy, and the only traceable origin was to a tuberculated leper living next door. The island of Molokai is used as a leper asylum, where all the lepers of the Hawaiian Archipelago are sent. A Catholic priest, Father Damen, devoted himself to their service, lived among them some years, and eventually became a leper, the disease commencing in the leg and ear. The fact that leprosy commences most frequently on exposed parts, especially the hands and feet, encourages the idea of contagion from outside, as may have taken place accidentally in the above and similar cases. Lastly, Damsch[1] appears to have proved that leprosy may be communicated to animals

by inoculation. It is stated by this experimentalist that portions of leprous tissue containing cells or bacilli remain intact in the bodies of cats and rabbits for months, but when they penetrate processes occur analogous to leprosy in man. As with cholera so with leprosy—it has been argued that it is only contagious in its endemic area. Thus Ghose [2] states that if leprosy is introduced where the hygienic conditions are good, the disease will not spread; and Kaposi [3] refers to the facts of persons having become affected after taking up their residence in leprous districts; while, on the other hand, leprosy dies out of families when they move to districts in which the disease does not exist.

The inquiry into the questions of heredity and contagion is, however, almost hopelessly confused by the fact that when inquiry is directed to heredity it is impossible to exclude the factors of contagion, or the influence of a common exposure to some noxious agent. When the inquiry is directed to contagion it is impossible to exclude the probable influence of heredity. The numerous statistics which have been collected by various authors show a wide divergence of results, and are not worthy of confidence. The evidence which may be brought forward in favour of heredity, and that which may be brought forward in favour of contagion, appear to be equally conclusive and equally liable to objection. My own opinion is that leprosy is both propagated by hereditary taint and by contagion. The fact of leper families abounding in India cannot be ignored, notwithstanding that, as previously mentioned, some of the children of lepers may escape the disease. Similarly, the fact of persons becoming the subject of leprosy without belonging to leper families cannot be ignored, and this must be attributed to other than hereditary taint. As there are various other diseases which are admittedly hereditary, but which do not affect the whole

[1] Virchow's *Archiv*, April, 1883.
[2] *Himalayan India.* [3] *Ed. Med. Jour.*, 1883.

of a family, so we have no right to assume that leprosy is not hereditary, because some members of a leper family escape. Similarly, we have no right to assume that because so many escape the disease it is not contagious; for even as regards the most contagious of maladies, immunity of the majority and affection of the minority is the rule. We have in syphilis an instance of a disease which may be both contagious and hereditary, and this I believe to be the case with leprosy. I do not, however, hold that leprosy is communicated by infection, *i.e.* through the medium of the atmosphere, as certain zymotic diseases are communicated. And I think that the propagation of leprosy must take place by contagion through the abraded skin. This would explain the reason why so many escape, although in constant communication with lepers, whereas accidental contact of leprosy poison with the abraded skin would explain such cases as that of Dr. Atkinson, referred to at p. 389. Moreover, I believe that I have so traced the communication of leprosy to an Indian female, who was in the habit of attending to lepers at the Joudpoor dispensary, who became the subject of leprosy, and in whose family no disease of the kind was apparent. That unknown quantity, malaria, has also been regarded as the cause of leprosy; *first*, because leprosy is very frequently found in so-called malarious countries and in malarious localities; *secondly*, because as in Great Britain leprosy has declined as drainage became general; and *thirdly*, because periodic febrile manifestations not unfrequently accompany the development of the different stages of leprosy. It may, however, be remarked that there is scarcely any disease, from epidemic cholera to epilepsy, from sunstroke to dyspepsia, which has not been referred to malaria; that leprosy occurs in cold northern climates where there are no malarious maladies and assumedly no malaria; that the decline of leprosy as drainage becomes general cannot be attributed to that one cause, the progress of drainage being always accompanied

by progress in other respects, by the advance of civilisation, implying better water, better ventilation, better food, and better general sanitation and personal hygiene, all of which tend to diminish most forms of disease; lastly, that periodical febrile manifestations accompany the progress of other diseases, of which elephantiasis and syphilis are examples. Fond as we are of attributing diseases to malaria, I think we must acquit this unknown agent of being the cause of leprosy. In short, I agree with Carter, who states that 'neither anæmia, scurvy, malarious fever, nor skin diseases have shown necessary co-relation.'

As regards *unsanitary conditions*, leprosy, like other diseases, may flourish under such circumstances, insomuch as they entail a state of human system below par, and therefore predispose to any malady, but there is no evidence that bad hygienic conditions can cause leprosy. Whatever induces cachexia, as defective diet, poverty, filth, damp, scurvy, impure water, exposure, will, like syphilis, predispose to leprosy; hence its common prevalence among the lower classes in India.

A form of bacillus has been found in the tubercles of leprosy by Hansil, Cormel, and others, and the lymphatics have been supposed to be the channel of infection. We cannot, however, accept as proved that the so-called *Bacillus leprœ* is the infecting agent. Carter has shown that the bacillus of leprosy and that of tubercle so closely resemble each other as to lead to the inference that they are the same. The bacillus of leprosy is best studied in the serum, squeezed out of tubercles by an ordinary pile clamp,[1] and when stained the bacilli may be recognised with a one-fifth or one-seventh objective. Neve gives the following recipe for staining fluid: Magenta crystal, one drachm; methyl blue, thirty grains; aniline oil, 100 minims; rect. spirit, one ounce; water, one ounce. And he believes leprosy

[1] Neve, 'Diagnosis of Leprosy,' *Ind. Med. Gaz.*, June, 1885.

contagious by the bacilli. Neve also regards the presence of the bacilli as diagnostic of leprosy, but, as previously observed, the bacillus of leprosy is exactly like that of tubercle.

Leprosy has also been regarded as a form of scrofula, but in India the two diseases do not seem allied.

Morehead was of opinion that elephantiasis and leprosy were allied, with this difference, that in the latter there is more general and extensive exudation-deposit, and a greater deviation in it from the healthy blood-plasma as shown by its readiness to undergo softening and ulceration.

The *connection* between *syphilis* and *leprosy* has long been a disputed point. Carter is of opinion there is no evidence of such connection, leprosy 'often occurring in persons whom it would be a mere presumption to tax with syphilis or its sequelæ.' But we are aware that syphilis, or its sequelæ, sometimes presents in persons who do not know they have it, and who also would not be suspected of contracting it immorally. As further proof that the diseases cannot be the same, it is stated that persons with leprosy may contract syphilis. But even a Hunterian chancre may be contracted twice, although the probability is against such re-infection, as it is, I believe, against the contraction of syphilis by the leper, which I have never seen. On the other hand, there are points of resemblance which lend colour to the idea of identity. Like syphilis, leprosy possesses affinities to the more active zymotic poisons, insomuch that it not unfrequently developes as an acute febrile eruption developes. Again, the sores of leprosy and the sores of syphilis have in some respects a visible resemblance. Hoarseness and nasal voice are often symptoms of leprosy, and sometimes the nasal and palate bones are destroyed as in syphilis. The manner in which syphilis induces nervous affections is also well understood, and such affections are a marked feature in leprosy. Neither would it appear that the tubercles of

leprosy and the deposits of syphilis are very different in character, for they have a very close pathological resemblance.

Amongst the protean forms in which syphilis manifests itself, there are many more extraordinary than anything appearing in the course of leprosy. Simon and Lindworm long since asserted the connection of the two maladies, regarding leprosy as the offspring of syphilis; and other writers have described what they rightly termed 'syphilitic leprosy.' Others, while denying a more intimate connection, have admitted that the syphilitic taint, inherited or otherwise, will prepare the system for leprosy. I have long considered leprosy to be a form of inherited syphilis, which, like other inherited taints, is partial in its distribution, and may not develope in one generation, although it may do so in the next; the development being determined by some condition of constitution or temperament with which we are unacquainted, aided by favourable surrounding circumstances.

The classification of the different varieties of leprosy as adopted by Carter is as follows:—The species he regards as *lepra arabum*; the varieties as *lepra leprosa* (synonym: lepra maculosa), *lepra nervosa* (synonym: lepra anæsthetica), *lepra nodosa* (synonym: lepra tuberculosa): the first variety being the eruptive condition, the second the nerve disease, and the third the nodular skin disease.

Symptoms.—Leprosy is frequently preceded by constitutional symptoms, the principal being languor and depression, deep-seated rheumatic pains, dyspepsia and febrile attacks. Usually, as Carter[1] observes, the disease progresses by slow movements, and creeps over the superficies of the human frame at an almost imperceptible rate. But an acute leprosy has been noticed by Danielsen and Boeck, which, however, Carter has not seen as a primary malady, although aggravations do so occur. It is probable the acute leprosy

[1] *Report on Leprosy*, 1867.

of the authors mentioned answers to the febrile condition, sometimes ushering in or aggravating leprosy in India; for there is an essential and apparently specific form of fever belonging to the initiation and aggravation of leprosy.

Either with or without the constitutional symptoms referred to above, two main symptoms manifest themselves, viz., hyperæmia of some part of the skin, and defective sensibility of the peripheral branches of the spinal nerves. The hyperæmia of the skin often takes the form of diffused redness (on the head or face), or of circular spots or blotches of irregular size. Such spots may be found on the trunk or limbs, also on the face, neck, hands, or feet. They are tender, irregular, and elevated, varying in size from a few lines to several inches. They are of a dull, coppery, or purplish tint, which, when it fades, leaves a pigmentary stain. The skin, so affected, is elevated, from serous infiltration, but at a more advanced stage it becomes slightly depressed in the centre, with a well-defined or elevated margin. Finally, the pigment more or less disappears from the centre. During the changes which occur the patches are sometimes bronzed, to which the term *morphœa nigra* has been applied; sometimes red, *morphœa rubra*; at others white, *morphœa alba*. These spots, however, are not true morphæa, which is a special affection of the skin, and has nothing in common with leprosy. Of whatever colour, the patches are usually glistening, devoid of hairs, which fall off, and they do not perspire. In all these cases there is a certain degree of numbness, although it may not be complained of, but there is little or no pain. The skin loses its elasticity, becoming dry, hard, and horny.

This condition of *lepra maculosa* may remain unchanged for an indefinite period, but eventually one of the other forms develope, or such forms may develope without the preceding maculosa.

Sometimes the disease is principally confined to the ner-

vous system, and then as the primary symptoms there is loss of sensation in some part of the surface (*Lepra anæsthetica*). The loss of sensation may be in patches as above noted, or it may be on the face or on parts of the body, or of the fingers, toes, hands, or feet. Sometimes the loss of sensation is so confirmed that flame does not cause pain, and so extremities have been burned while cooking, or gnawn by rats while the person slept. Occasionally the loss of sensation is preceded by tingling, burning, or exalted sensibility, which gradually gives place to numbness. Sometimes parts which were anæsthetic become highly sensitive. Occasionally the motor nerves become affected, resulting in tremblings and jerkings, wasting of the limbs, or even paralysis. Deep-seated pains in the bones or joints, worse at night, are accompaniments; also much progressive exhaustion and debility. There is great proneness to the formation of bullæ, generally on the patches if they exist, which burst in a few days, leaving an oval or round ulcer, with a punched-out appearance.

When leprosy assumes the tubercular form the conspicuous symptom is the gradual growth of solid papules or nodules, or tubercles in the skin from the size of a pin's head to that of a walnut, causing the face, or other part affected, to assume a curious nodulated appearance. These formations are hard, of a brownish colour, there is little or no pain from them, and they often increase with attacks of fever, remaining stationary in the intervals. After a time they undergo softening and ulcerate, or watery blisters appear, which afterwards become ulcers; these ulcers progress slowly downwards, sometimes destroying the bones beneath, sometimes healing with a depressed cicatrix. Oftentimes the fingers and toes are first attacked, either with anæsthesia or tubercular growth. But ulceration is not restrained to cases where this occurs. First, there may be simply general swelling of the part, such as the toe, fingers, heel, the disease

appearing to have an internal origin; then ulcers form; or the nails become black, shell off, and leave unhealthy ulcers. Sometimes the disease appears to commence by absorption of the articulating surfaces of the joints of the toes or fingers, which may end in ankylosis or in spontaneous dry amputation or *ainhum*. When there is ulceration without any, or the very least discharge, the extremities appear to dry gradually away. Sometimes the fingers and toes, instead of ulcerating after swelling, then shrink away, leaving the joints prominent (lepra nodosa). Whatever occurs to the fingers and toes, the forearm and leg become more or less wasted, while the local nerves, especially the ulna, become nodular from the first large joint downwards. The disease may now remain stationary for an indefinite period, or the ulcers may heal and recovery appear in prospect. But ultimately ulceration of some part recurs, and frequently the fingers and toes become contracted, or are totally lost from sloughing ulceration. This form of leprosy is often spoken of as *lepra mutilans*, and is the condition most usually seen in Indian leper asylums. The destruction never extends beyond the metacarpal or metatarsal bones, probably in consequence of the greater vitality of the parts nearer the trunk offering a more effectual barrier. The higher joints may present effusion, but suppuration does not occur. This form of leprosy, loathsome as it is, is less to be dreaded as regards the sufferings entailed than many other diseases. The liability to ulceration and caries is in inverse ratio to the sensibility of the part. Hence with these lesions there is often little suffering, and the ulcers may be treated with strong caustic without producing pain.

The condition spoken of as *ainhum*, mentioned above, was described by Lima of Bahia in 1867, and said to be peculiar to the African race. A groove or furrow forms at the base of the little toe inferiorly, afterwards extending to the whole circumference, becoming deeper till the toe is

attached by a pedicle, only brought into view by separating the walls of the furrow. The distal portion swells and appears as a round knob; this furrow is caused by a constricting band of hardened and contracted skin, or a local *sclero-derma*, leading to faulty nutrition of the parts beyond, and probably bone or articular disease. The cause is said to be obscure, but I have several times seen this in connection with leprosy. There is reason to believe the condition may result as the only manifestation of leprosy, and that it may be excited by an injury and be connected with caries of the phalanges. Assistant-Surgeon K. B. Cooper, Bombay, has recently reported an instance of the kind, and he considers it occurs in natives in consequence of their habit of going about bare-footed exposing the toes to injury.

Long before the period of ulcerations referred to, the constitution is undermined, and there is great physical debility. Now the mucous membranes also become affected. Hæmorrhages from the nose, mouth, and throat may occur, and there is much thickening, tubercular deposit, or ulceration apparent in these structures, followed by destruction of bone, not unlike the ravages of tertiary syphilis. The fatal termination is by lung-congestion, by diarrhœa or dysentery, by melæna, or the person dies exhausted with preceding delirium. The fact of metastasis sometimes taking place from the leg to the arm or *vice versâ*, should be noted. Also that the parts affected occasionally assume the appearance of erysipelas, or are really attacked by erysipelas.

Although typical cases of the different varieties of leprosy (*maculosa*, *anæsthetica*, *tuberculosa*) are met with, although Carter states the anæsthetic variety is most prevalent in India near the sea-coasts, and although the natives themselves understand the difference (*sūn* or *sūnbahiri* being numbness or nerve-leprosy, *kod* tubercular leprosy, and *pat-qurmi* or *raght-pite* the mixed form), I believe a mixed

form is much the most common. There is always both a nerve and skin affection present, the latter resulting from the former.

It is a curious fact that wounds heal remarkably quickly in the leper. This was well exemplified in the person of a farrier in the Bombay cavalry, who had his testicles and penis bitten severely by a horse. Skin diseases such as psoriaris or itch, which are often co-existent with leprosy, are intractable. Khory states, 'The natives of India look upon the malady as a punishment from God, and regard it with great awe and superstition.' But according to some authors lepers are almost invariably contented and happy. This, however, only applies in India to the earlier stages of the disease. In most parts of the country so long as the leper can work he is no outcast from society; his fellows live in the same house with him, partake of the same food, and even allow him to marry. If the malady is chronic and does not entail great suffering, I believe many lepers would rather endure the disease which enables them to obtain a living as mendicants than be free from it on the condition of working. Thus many, while taking advantage of leper asylums during the wet weather, prefer their liberty and the precarious existence of begging during the remainder of the year. When the disease progresses and they suffer from pain and exhaustion, they become morose, inclined to brood in solitude, drink spirits, eat opium, and smoke gunjah; in fact, appear to endeavour to shun a sense of their unfortunate condition. Formerly, before it was put a stop to by the British Government, *sumajh*, or burial of lepers alive, was a general practice, and always with the consent of the leper himself, who, frequently declaring to his relatives he was tired of life, would ask them to perform *sumajh*. The burial of lepers alive has been performed within my own knowledge in the native states.

The average duration of the disease is, according to Boeck and Danielsen, eighteen years and a half, but in India it would appear to run a much quicker course.

Post-mortem appearances.—Lungs often diseased with tubercular deposits. Liver and spleen generally enlarged and softened. Deposits about the spinal cord, which has been found hardened. Thickening and deposits about the membranes of the brain. Nerves leading to the parts affected swollen. Sheaths of cutaneous nerves thickened and distended with exudative deposits. Virchow states, the pathological element of leprosy differs in no essential respect from that of the gummata of syphilis.

Diagnosis.—The advanced stage of leprosy cannot be mistaken by a person acquainted with the disease. It is stated leprosy may be diagnosed in the earlier stages by taking the patient into a dark room and passing a blue flame before the face. If leprosy is present, the skin assumes a reddish hue. If there is a greenish hue, there is no leprosy. This test is said to be much used in China. Neve, as before remarked, regards the bacillus as distinctive. A deviation of the palatine arches from anæsthesia, thickness of speech, affection of the gums resembling scurvy, anæsthesia of the little finger, atrophy of the ball of the thumb, a thickened and pendulous condition of the ear-lobes or of the eyelids, a chronic conjunctivitis, have all been regarded as characteristic of the earlier stages, but none are of constant occurrence. In simple leucoderma, with which the first stage is likely to be confounded, there is no anæsthesia or elevation of the white spots, no enlargement of the nerves, no conjunctival affection, or thickening of the ear-lobes, or other signs of leprosy. Lepra tuberculosa must be distinguished from the malady known as *Fibroma* characterised by the growth of a multitude of tumours from the skin (*vide* Skin Diseases). Chevers remarks that as a Hindu leper cannot inherit estate, because as one impure he is debarred from the

sacred duty of firing the funeral pyre, the differentiation of leprosy becomes doubly important.

Rheumatism in its sub-acute form often complicates leprosy. The deep-seated pains of the earlier stages of leprosy may be mistaken for mere rheumatism. On the other hand, sciatica or lumbago may be regarded as the pains of leprosy when they complicate the latter disorder.

Treatment.—This is vague and unsatisfactory. *Chalmogra* oil outwardly and inwardly is perhaps of the most decided utility, and cures have been described as effected by this means. The oil is taken in 6 to 12 minim doses. *Kowti* oil has also been stated to be useful. Other oils, such as bawarchi oil, ground nut oil, cashew nut oil, gurgun oil, particularly the latter with lime-water in the proportion of 2 to 4 drachms, have all been lauded, gurgun oil being found most useful when there is a squamous or eczematous condition of the skin. But in many cases arsenic and iron seem to have most effect on the disease, by strengthening the constitution. Medicated baths have been much recommended. In the tubercular variety fumigation by means of vaporised carbolic acid has seemed useful. But it must be recollected that the disease has a chronic course by exacerbations, with intervals of apparent progress towards health, or of quiescence, and reported cures must be received with caution. In every assemblage of lepers some will be found who no longer suffer from the disease in its active condition.

The ulcers and sores require chiefly stimulating applications with perfect cleanliness.

Amputation except for the removal of sphacelating parts is inadmissible, as the diseased condition almost always returns in the stump, and if not, elsewhere. Good nourishing diet with a due proportion of fresh vegetables is most important. This appears to have been well understood in former times, for the records of ancient English lazar-houses give a dietary which for those days appears luxurious. Re-

moval from the locality where the disease was apparently contracted is always advisable, but if benefit is to be obtained change should be effected early; for lepers who in an advanced stage of the malady ramble through the country, are rarely improved. It is doubtful, however, if leprosy has been cured by any means. When amendment occurs under the influence of tonics, oils, nourishing diet, good hygiene, and change of air, the cachectic leper becomes, as Chevers observes, a robust leper, but he remains a leper still.

Munro, Carter, and others have proposed the establishment of leper villages, and the segregation of all lepers: a desirable but impracticable procedure, both from the expense it would entail, and from the fact that force only would confine Indian lepers within the prescribed bounds. The diminution of leprosy is rather to be sought for by the progress of sanitation generally, in which I include the cheapening of salt, the supply of pure water, the prevention of scarcity, and measures against the spread of syphilis.

CHAPTER XXIII.

LIVER DISEASE.

Position of the Liver.—Endeavours have been made to correspond the area of hepatic dulness with the height of the individual. Thus it has been stated that in a person 5 ft. 7 in. the dulness should extend downwards four inches; in a person 6 ft. high four and a-half inches; but such measurements are not reliable. The healthy liver reaches as high as the fifth rib or cartilage of the fourth rib, dulness commencing in a direct line downwards from two inches below the right nipple. The inferior border comes down as low as the lower margin of the ribs, or even slightly lower. To determine the upper border correctly the stroke should be strong, as superiorly the lung descends between the ribs and that portion of the convex surface of the liver which fits into the diaphragm, and it requires strong percussion to bring out the dulness. To determine the lower border correctly the stroke should be slighter, for if too strong we obtain the sounds of surrounding hollow viscera, through the thin layer of the liver, which covers them before we arrive at its margin. The left confine of dull hepatic area terminates at a point about one and a-half inches to the left of the lower margin of the xiphoid cartilage, but the presence of a distended stomach may mask the dull sound from the thin left margin of the liver. Superiorly also the heart lies so near the liver that the flat sound of one cannot be distinguished with accuracy from the flat sound of the

other. But the size of the left lobe is subject to great variation. Frequently, when short or compressed, it extends but slightly into the left hypochondrium; when large it may stretch over the pyloric orifice and first portion of the duodenum as far as the spleen. In the median line, the lower edge lies usually a little above the middle of a line connecting the point of the xiphoid cartilage with the navel. At the right side dulness, commencing in the seventh intercostal space, extends to the tenth, or even to the eleventh. Posteriorily it is on a level with the tenth or eleventh rib, more rarely with the ninth; but the inferior border dulness being continuous with that of the right kidney, cannot be determined. From the foregoing it is evident that the size of the liver varies a good deal even in health. In women who wear tight stays it may project downwards an inch or more lower. It is also lower in those who habitually take much horse or camel riding exercise. In the upright posture it may be lower than when the person lies on the back, especially in some people having naturally long or lax ligaments; hence the desirability of examining the organ in both positions. It should also be recollected that a full stomach and a distended colon push the liver downwards and backwards; that fæcal matter in the colon, or abdominal tumours, may be mistaken for hepatic dulness; that a distended gall-bladder may cause a strictly defined dulness, lower than the dulness of the liver; and that considerable disorganisation of the liver may exist without influencing its position, or dimensions, or sounds on percussion.

Measurements taken when the liver is removed from the body are useless, as the whole organ becomes flatter and broader. As with the *size*, so the *weight* of the liver varies, the average being $4\frac{1}{2}$ lbs. in the adult. One-twentieth of the weight of the whole body in children to one-fiftieth in old age may be accepted as an approximation.

Although the liver may be felt with ease, particularly if the patient turns towards the left, still percussion is preferable for the examination of the organ, for its limits can then be obtained with some precision, even when too tender to admit of manipulation. For examination by percussion the person should lie straight, and if possible afterwards stand. For examination by manipulation he should lie with his shoulders well raised and the knees well bent, inclining towards the left side. If in this position the fingers are bent, and the points pushed upwards and backwards under the liver, the pressure will, if the organ is inflamed, produce pain. If this does not cause pain, but pressure directly backwards does do so, it is probable the colon is the organ implicated.

Structure and Functions of the Liver—It will be recollected that the liver receives its blood from the portal vein and hepatic artery, the blood being returned to the vena cava by the hepatic veins. The portal vein, hepatic artery, and hepatic duct run in company invested by Glisson's capsule, surrounding and enveloping the lobules. The structure of the liver differs from that of other glands, the structural units, the acini, not being enclosed by a well-marked layer of connective tissue, but being grouped together in immediate contact, excepting for the ramifications of the vessels and duct.

The peculiar manner in which the liver is supplied with blood shows that it must serve some principal purpose, probably direct purification, with regard to the stream arriving by the portal vein, which has just gathered up various material in its course through the digestive organs; and the frequency with which metallic poisons are intercepted, or retained in the liver substance, may be adduced as evidence.

Harley states if we compare our knowledge of the functions and physiology of the liver with that which the ancients attained to, it will be found that by reasoning they deduced that the liver secreted bile and was a primary

organ of sanguification, which is all we know, although in a more definite and precise manner. But I venture to say we do not yet know how this takes place in any definite or precise manner. We know that the bile is the first digestive agent with which the food in the shape of chyme comes into contact after leaving the stomach, the two forming a white flocculent emulsion. Physiologists also tell us that the saccharine and albuminous materials are taken up by the capillaries of the portal vein, and carried at once to the hepatic cells, where they are converted into glucogen and sugar, and probably into urea, subsequently eliminated by the kidneys. When this function (the transformation of albumen into glucogen and the latter into sugar), or at least the saccharine portion of it, is interrupted, the hepatic cells are filled with glucogenous albuminoid material, and amyloid or albuminoid liver is the result. When the saccharine function is too active the excess is excreted by the kidneys, and glycosuria results. The fatty and oleaginous materials being absorbed by the lacteals, are carried by the thoracic duct into the general circulation. But as others believe they are previously acted on in some manner by the bile, a portion of which is absorbed with them; the precise manner in which, and when, the liver acts on fatty and oleaginous atoms is, however, a mystery. All that we know is that the liver—at least when in an abnormal condition—has some action on the fatty elements, as is proved by the faculty the liver possesses of storing them in excess, forming fatty or lardaceous liver—a faculty, however, shared by many other structures, both glandular and otherwise. It is also believed that the fatty elements are transformed into cholesterin, the basis of gall-stone. Then there is the calorifying function. As is well known, every chemical change is associated with active oxidation, which produces heat. From the number and variety of the chemico-physiological processes, which are constantly taking place in the

liver, the organ is the warmest in the body, and contributes greatly to the maintenance of animal temperature. It may be perhaps correct to describe the different functions of the liver as sugar-manufacturing, fat-modifying, calorifying and excretive. But as it is also ascertained that saliva acts on starchy matters, gastric juice on albuminoid matters, pancreatic juice on fatty matters, all that can certainly be advanced is that the principal use of the liver is to *produce* bile, which is partly used in assisting the emulsion and absorption of food, and partly in the conveyance of excretory matters from the system, when it also acts as an antifermentative and as a natural aperient.

Neither does the *appearance* or *chemistry* of the bile help us. Fresh human bile is of a brownish-yellow colour, but varying according to concentration and the food taken.

It is composed of *biliverdin*, derived probably from the colouring matter of the blood; of glycocholic and taurocholic acids, of which little is known; of cholesterin, a pure white fatty matter which forms the basis of gall-stones, and is said to exist in the blood; of hepatic resin, of sugar, and of soda, potash, and iron. The amount of bile secreted is supposed to be nearly two pounds daily, and we are sure some of it passes out of the system, rendering the liver both an excreting and a producing organ.

Varieties of Disease.—It was at one time the vulgar custom to refer nearly all diseases to the liver, and it may now be asserted that when this organ is disordered, the whole system suffers in a greater degree than from a disturbed state of almost any other organ. Others, especially specialists, have referred to different organs maladies which should be chiefly attributed to a faulty liver. The stomach and bowels especially often present symptoms which careful investigation discovers to have their seat and origin in the liver. Every form of liver disease is met with in India, but there are some much more common than others.

Murchison objected to the usual classification of liver diseases as adopted by Morehead, Budd, &c., on the ground that the functional disorders specified do not agree with what we know of the functions of the organ, which he summarised under three heads, viz., *formation of glycogen*, which contributes to animal heat, and to the formation of urea; the *metamorphosis of albuminoid matter*, and production of urea, and other nitrogenous products; *the secretion of bile*, which assists in the conversion of fat, &c. And under this summary Murchison named no less than nine functional derangements. I am of opinion, however, that it is not practically possible to diagnose or distinguish these functional derangements even if they exist independently of each other. It is not possible to distinguish disorder of metabolic functions, for instance, from disorder of biliary function, for the one will not exist without the other. The most convenient classification, and the one most in accordance with what we meet with in actual practice (in India) is I believe as follows: 1st.—*Functional.* *A.* Increased secretion of bile. *B.* Deficient secretion of bile, both necessarily attended with a more or less congested state of the liver, often leading gradually to 2nd. *Organic.* *A.* Chronic congestion. *B.* Acute congestion or inflammation. *C.* Abscess. The liver diseases *ordinarily* met with in the East are comparatively not very diversified, most of the some five and twenty hepatic maladies described by systematic authors being comparatively rare and presenting nothing special.

Increased Secretion of Bile.—The system of the European exposed to the high range of temperature of India, immediately begins to accommodate itself to the altered circumstances. The rarefied air probably containing less oxygen than an equal volume of the atmosphere of a colder climate, prohibits the elimination by the lungs of the same amount of carbon previously expired, while perhaps more of the latter in the shape of food and drink is daily introduced into the

system. At the same time less exercise being perhaps taken, there is diminished combustion and comsumption of tissue. As a relief to this condition, the liver is excited to a temporarily increased action, and an increased, often copious and acrid, secretion of bile takes place, which may manifest itself by an irritating diarrhœa. Instead of passing off by the bowels, the bilious material in some cases producing nausea, sickness, and headache, eventually causes the person to suffer from what is popularly known as a bilious attack, or a bilious colic. This (almost the only process of climatisation, if it may be so called, which takes place in India) will in most cases subside after the bilious material has passed away. Sometimes the aid of an aperient salt is required, and less frequently it is desirable to allay the intestinal commotion which the acrid bile excites by small doses of opium or the compound ipecac powder. In all cases moderation in diet is necessary, and in severe cases the recumbent posture should be insisted upon, and spoon diet only allowed.

Deficient Secretion of Bile, or Torpor of the Liver.—As in other organs temporary excitement is followed by depression, so this excessive action of the liver is followed by more or less torpor, leading to deficient secretion of bile, costiveness, and pale stools, and giving rise to headache, furred tongue, foul taste, and viscid mucus in the mouth and fauces, especially in the morning, and an irritable, excitable, and depressed condition. The faulty function being connected with albuminoid disintegration, and the non-conversion of albuminoid matter into urea, in long-continued cases the condition described by Murchison as *lithæmia* becomes established, marked by an increase of dyspeptic symptoms, and the production of lithates and lithic acid, tending towards skin maladies, biliary calculi, and gout. Podophyllin, euonymin, iridin, taraxacum, and saline aperients, exercise, and moderation are here demanded. In some cases hepatic derangement and diarrhœa are less manifest, and the system is relieved by

a succession of painful boils, or by excessive eruptions of lichen tropicus.

Congestion, Chronic.—Should neither of these modes of relief occur, the liver becomes full, bulky, and perhaps tender, the condition of congestion being slowly but surely established. It is impossible to define when functional disturbance ends and organic changes commence, for the one is the origin and initiatory step of the latter. The primary condition appears to be a simple fulness, stretching, or turgescence from too much blood, which may arise from excess of eating and drinking, followed by the conduct into the liver of a large amount of carbonaceous material requiring a larger influx of blood into the organ for its metamorphosis. In addition to this as causes, there are sedentary habits and insufficient exercise; too much sleep, during which the excretion of carbonic acid is considerably diminished, the heat of the climate, and consequently expanded air and diminished oxygen in a given bulk of atmosphere, exposure to chills and damp, imprudent use of the cold bath, or of punkahs and tatties, the changes of temperature occurring from the sea-breeze near the coasts and during the rainy and cold seasons, crowded barracks, or unventilated dwellings, and the cold stages of paroxysmal fevers. These causes when acting singly are frequently insufficient of themselves to produce disease, although they dispose to it; but when several are combined their influence is more certain, and after they have been some time in action, the operation of some additional accident is followed by the full effect, terminating if not relieved in organic change of the hepatic structure. Thus we have sub-acute hyperæmia or congestion, followed by permanent enlargement of the liver, with increase of cell growth and connective tissue, with or without degeneration of liver cells.

It is not intended to consider that passive hyperæmia of the liver which arises in every climate from mechanical obstruction to the flow of blood through the hepatic vein

and inferior vena cava; from organic disease of the heart, lung affections, &c.

The *symptoms* of ordinary chronic congestion of the liver come on very gradually, and are usually preceded by more or less of the functional disturbances already described, which are gradually attended with more decided dyspeptic symptoms. There is depression of spirits, defective appetite, coated tongue, bad taste in the mouth, accumulation of mucus about the fauces, frontal headache, irregular action of the bowels which are often constipated, stools varying in colour, nausea or perhaps vomiting in the morning, a sense of fulness or weight in the right side, with perhaps epigastric tenderness, recumbent posture generally uneasy on the left side but sometimes on the right, pulse slow, oppressed, and sometimes irregular, urine depositing lithates. As the disease progresses the countenance becomes sallow. The condition is in fact an exaggeration of that described as deficient secretion of bile; the latter indeed being the first link in the chain of sequence.

If the condition is not relieved, either purulent suppuration or some other enlargement of the liver occurs, leading to further disturbance of nutrition, elimination, and disintegration, marked by increasing severity of the symptoms of impaired digestion and of derangement of the nervous, circulatory, respiratory, and urinary systems, and of the functions of the skin, which is usually dry and harsh.

The enlargement of the liver which occurs has been frequently regarded as chronic hypertrophy or inflammation, but after a time at least, if suppuration does not occur, there is something more, viz., the addition of fatty degeneration. When this is present, the enlargement is gradual, the liver descending below the ribs. But the enlargement is uniform, the border rounded, and usually without nodulation. The whole abdomen is also oppressed, and gives a 'doughy' feel to the fingers. There is also cough from pressure of the enlarged

liver on the lung. There is also an increasing cachexia, but as the secreting powers of the liver are to a great extent maintained, there is neither confirmed jaundice nor dropsy for an indefinite period, until such powers begin to be lost by increasing deposit, infiltration of, and pressure on the cells, when the atrophied condition and ascites may present, the latter from mechanical obstruction to the passage of blood (by the vena cava), which is pent up in the abdominal veins.

When the liver is fatty the deposit in slight cases is limited to the outer zones of the lobules, but in severe cases the whole is infiltrated, and thus the colour varies. A fatty liver, being full of oil-globules, floats in water, imparts a greenish stain to the knife, burns with a flame, is in fact in the same condition as the goose's liver after the bird has been kept in a dark room and filled to bursting every day.

Amyloid or *waxy* liver is not so often recognised as the result of tropical congestion as fatty liver, but it occurs as a sequel of long-continued cachexia. The structure of the organ is infiltrated with a peculiar homogeneous substance, which first affects the minute branches of the hepatic artery and then extends to the cells. The weight and size of the liver are greatly increased, although its form is not altered. It is hard and inelastic, and when cut the surface is grey or fawn yellow. Iodine changes the colour to red or brown, sulphuric acid gives a blue colour. When methyl-anilin is applied, the affected parts become violet red, the healthy structures violet blue. The symptoms are slow, with uniform enlargement of a dense, firm, resisting character. The lower border is round and regular, and there is neither pain nor tenderness, but simply a sense of fulness or tension. There is little tendency to obstruction of the portal circulation, and hence no ascites. The urine usually contains albumen. There is often troublesome diarrhœa, the stools being pale yellow. Anæmia is well marked.

In all cases where the liver becomes so much enlarged

as to descend below the ribs the fragility of its structure is much increased; it is more exposed to injury, and it is liable to rupture from slight causes. Rupture of the liver is not however necessarily fatal, unless some of the larger veins are involved.

Chronic Atrophy, or the cirrhoid or nutmeg liver, is not common among Europeans in India as in Europe. Livers are certainly found at post-mortems presenting very reduced bulk, adhesions, and thickening of Glisson's capsule, and the other characteristics of the cirrhoid state. But it is comparatively exceptional, and may perhaps be accounted for by so many succumbing to acute liver-affection, to the climate and manner of life tending more to induce fatty degeneration, and to the prevalence of anæmia, which in most countries is associated with amyloid enlargement. Cirrhosis is, however, much more common among natives than it used to be, owing to the more general consumption of spirits than was formerly the case. Cirrhosis is not, however, always traceable to alcoholic drinks, for it sometimes occurs in those who have not been addicted to drinking, as also in children. It may also follow acute hyperæmia of the liver, or 'interstitial hepatitis,' as it has been called; as if it were possible to diagnose, except from the after-effects, if an attack of hepatitis were interstitial or the reverse, which it is not. The symptoms of cirrhosis of the liver are not easily distinguished at first from ordinary indigestion. When, however, there is loss of flesh and strength, loss of appetite, pain or discomfort in the right side, following or not following an attack of congestion in a person who is regularly in the habit of drinking, commencing cirrhosis may be suspected. A little later this may be verified by the irregular margin of the liver being felt beneath the ribs. For as the liver contracts it assumes the well-known hobnail appearance. This contraction leads to narrowing of the blood-vessels which pass through the gland, and so causes the veins coming from the other organs

of the abdomen to be over-distended, resulting in hæmorrhoids or gastric congestion. Serum oozing through the intestinal veins causes diarrhœa which may end as dysentery; or ascites, which may impede the thoracic organs. Epistaxis, or even apoplexy may result; the latter, if there is an atheromatous state of cerebral arteries, which may be unable to stand the strain on the arterial system from impediment to free circulation of venous blood in the liver.

The *treatment* of chronic congestion of the liver is not satisfactory. An occasional podophyllin purge, followed by the use of Friedrichshall, Hunyadi Janos waters, or Carlsbad salts, is recommended, with the use of mustard leaves or iodine paint over the liver as often as can be borne. When podophyllin gripes, and does not agree, as is sometimes the case, a mercurial purge, as five or more grains of blue pill, should be substituted, or euonymin or iridin may be tried. Care in diet and abstinence from alcoholic liquors are essential. Exercise, especially horse exercise, to the verge of fatigue is usually advisable. Change of climate from the tropics is the only radical remedy, but this must be taken in time before organic changes have made much progress.

When the liver is enlarged, and there is reason to suppose it fatty, oleaginous matter, sugar, and sugar-forming foods such as belong to the farinaceous groups, should be avoided as much as possible. Exercise should be taken short of fatigue, mineral waters, as Hunyadi Janos, may be used, and carbonate of ammonia in five-grain doses may be tried. When the liver is both enlarged and hard (hypertrophic cirrhosis) Harley advises puncture of the capsule in several places, with a small trocar to relieve pressure.

Acute Congestion and Hepatitis.—It is unnecessary to make a distinction between these conditions, the one being an aggravated form of the other. The causes are the same, and the immediate exciting cause may generally be traced to exposure or chill. Acute congestion or hepatitis is

chiefly a malady of new arrivals in the tropics. In older residents it occurs more gradually with less febrile disturbance. European females are less subject than males, and in children it is uncommon. Among the better classes of Europeans, who are careful in their habits, the disease is not so frequent as formerly, when habits of life were different. It is, however, very frequent among natives.

Symptoms.—Pain in the right hypochondrium, increased by pressure, by deep inspiration, by sighing, by lying on the left side; frequently a sense of aching or dragging; pain in the right shoulder, sometimes under the right clavicle, or occasionally in the left shoulder; deficiency or redundancy of bile in the evacuations; high-coloured urine; yellowness of conjunctivæ, and exceptionally jaundice; breathing a little oppressed, and pulse quickened, but often not much so; temperature increased from two to three degrees; tongue coated grey, with red projecting papillæ. But all symptoms vary much in accordance with: first, the extent of the liver which may be affected, and secondly, with the part of the liver implicated. Thus, when the concave surface of the liver is affected, pain is more obscure and referred to the back, but the functions of the stomach are more disturbed, there being vomiting, hiccup, and epigastric pain, and the stools contain less bile—indications of the duct, duodenum, and pylorus being affected. Tension of the abdominal recti muscles is indication of the anterior part of the liver being implicated. Pain of an acute character, with much febrile disturbance, leads to the conclusion that the peritoneal covering is more involved; disturbance of the functions of the kidneys, irritable bladder, and lumbar pain, that the posterior part is most affected. Pain is always more or less severe, according as the congestion is seated in the substance of the liver or towards the circumference, and in some instances of the former it is little felt. The amount of febrile disturbance is always less marked in asthenic

persons than in the robust, and in the former it is often remittent. When the convex surface of the liver is affected there is usually shoulder pain, and the peritoneum and under surface of the diaphragm are also affected, with often considerable congestion of the lung lying immediately above. In cases of this description there is generally much febrile disturbance, and the distress of breathing is very marked, being carried on chiefly by the intercostals, while there is a sense of tension across the chest, with suppressed cough, and inability to take a deep inspiration. Melœna may occur in advanced cases. Shoulder pain is not worthy of great confidence, unless combined with other evidence, and increased by pressure on the liver. It has been attributed to the anastomosis of a twig of the supra-clavicular nerve with the pneumogastric, or from connection between the sub-clavian and phrenic. It may be due to rheumatism when it is increased by movement; but this is an uncertain characteristic, the pain often being increased by motion when there is no rheumatism. When this shoulder pain has been severe during hepatitis it often disappears or becomes intermittent if suppuration occurs.

In the enumeration of symptoms it is stated there may be *costiveness* or *diarrhœa*, *deficiency* or *redundancy of bile* in the evacuations. As a general rule a redundancy or at least the normal quantity of bile is present. That an inflamed or irritated organ will secrete an additional proportion of its especial secretion is evidenced by the increase of mucus from inflamed surfaces, and by experiments on organs, as the pancreas for instance, which when irritated has been found to secrete many times the normal amount of pancreatic juice. Secretions from irritated or inflamed organs are also more or less altered in character, hence the dark and morbid appearance of the bile. But there comes a time when the inflammation is far advanced, and when no bile can be secreted from a disorganised structure, yet bile is still found

in the stools; such absence of marked change in the secretions being often due to a limited extent of the structural alteration, but in other cases, and probably to a greater degree, due to the secretion by the intestines of a material resembling bile. The continuance of bile in the stools has been explained on the theory that the hepatic or nutritive capillaries were alone implicated, the portal capillaries continuing their office; but I fail to see how inflammatory action can be so limited, when tissues are so closely connected. Deficiency of bile in the evacuations occurs when the tumefaction produced by the inflammation interferes especially with the gall-bladder and appurtenances. The passage of bile is then mechanically prevented, and is in such cases readily detected in the urine, which may be even muddy, and deposit brownish flakes. It is important that this condition should be recognised promptly, for the treatment requires to be varied (*vide* p. 419.)

The *state of the urine* during hepatitis varies very considerably. As before noted in the enumeration of the general symptoms, it is usually scanty and dark-coloured, and this from the presence of lithates. But, as mentioned above, when bile is not freely excreted the urine betrays its presence. From extension of the inflammation from the concave surface of the liver to the right kidney, albumen, and less frequently blood, may be found in the urine.

Diagnosis of Hepatitis.—There are several conditions which may possibly be mistaken for hepatitis, or *vice versâ*. When peri-hepatitis accompanies, and the upper surface is inflamed, while the pain is aggravated by deep inspiration, it may be mistaken for pleurisy, especially if an hepatic friction-sound is audible. But the pain of pleurisy is more stabbing and lancinating, and more aggravated by respiration than that from the liver, while the decubitis is generally less painful on the diseased than on the sound side during hepatitis, the reverse being usually observed in pleurisy.

There is ordinarily an absence of friction-sound unless the pleura is inflamed. From pneumonia hepatitis must be diagnosed by the absence of rusty expectoration and stethoscopic sounds of diseased lung, and by the enlarged or tender state of the liver. Also in pneumonia there is much difficulty in lying on the affected side. When a person with hepatitis lies on the left side there is increased uneasiness, and a dragging sensation. Only a careful examination of the peculiar symptoms presented will in some cases establish a correct diagnosis between hepatitis and gastritis. When the left lobe is inflamed symptoms very like gastritis present, the principal being continued vomiting, florid tongue, contracted pulse, and pinched face; but gastritis is rare, and the symptoms of hepatitis are, although similar, less intense than in inflammation of the stomach. As, however, with pleuritic inflammation and pneumonia so with gastritis, all may exist as the secondary result, and extension of the hepatic inflammation.

Post-mortem appearances.—It is not often that death occurs from congestion or inflammation alone, but a congested liver will be found large and dark from impeded circulation, and full of blood and bile. The texture is softened, and the colour varied according as the congestion is portal or general. There may be discoloration of the peritoneal covering, thickenings, and adhesions to the stomach, liver, and diaphragm. Or there may be deposits of coagulable lymph. The interior of the liver may show in some parts red patches, in others yellowish-coloured spots from the faintest haze to a deep line, or a multitude of light spots varying in size from that of a pin's point to a pea, and consisting of minute deposits which are rapidly becoming pustular. The gall-bladder may be enlarged and filled with viscid bile, or it may be contracted, with thickened coats. The serous coat of the portal vein is usually reddened.

Treatment.—In former days repeated venesection used

to be employed, followed by leeches over the liver or to the anus, but such measures are not now esteemed necessary. In fact, during the bleeding period it was argued that blood-letting ought not to be carried to the same extent in hepatitis as in other inflammations, owing to the peculiarity of the portal circulation, which was assigned as a reason of non-success, instead of the loss of blood itself. Salivation was also esteemed necessary, under the impression that if the gums were once touched the patient was secure from abscess, an idea which had no foundation in fact. Nevertheless, in former days I have seen persons recover speedily and thoroughly from hepatitis under moderate bleeding and slight mercurialisation. On the other hand, in later days I have seen persons recover equally satisfactorily without such measures. For inflammation of the liver occurring in robust persons it will be desirable in the first instance to unload the gall-bladder and bowels by a mercurial purge, which may be repeated as necessary. One or two doses of mercury at the commencement, or occasionally during the progress of the disease, will produce all the good that can be effected by this agent, and the benefit results from the removal of acrid bile contained in the ducts and gall-bladder, thus rendering the passage clear for the flow of newly secreted fluid which even during inflammation is often copiously formed. There is, however, one condition previously described when the action of mercury is of more importance than in ordinary cases. I refer to those instances where the inflammation attacks the concave surface of the liver, implicating more especially the gall-bladder and ducts. Without entering on the vexed question how mercury acts on the liver, it may be mentioned that it is an ascertained fact that it promotes the flow of the pancreatic juice. Now the pancreatic duct in perhaps the majority of instances opens into the bile-duct, forming the common duct. Hence the thin pancreatic juice must soften, dilute, and promote the flow of the bile when it is most

likely to be mischievously retained. Then, as soon as the bowels have been thoroughly moved, the tartar emetic treatment may be adopted. Two grains of tartar emetic are to be thoroughly mixed with two drachms of nitrate of potash, and the mass divided into eight powders, one to be taken in two ounces of water every hour. If this medicine, as is sometimes the case, acts on the bowels the repetition of the mercurial may not be required. If the tartar emetic produces too great nausea or vomiting, it may be reduced in quantity. These medicines promote both cutaneous and urinary secretion; they lessen the force of the circulation, and they render the blood alkaline. For less robust persons chloride of ammonium, as advised by Dr. Stewart, may be substituted for the tartar emetic mixture. Twenty grains should be given three times daily in water. When there is tendency to dysentery, Dover's powder eight grains, ipecacuanha one grain, and quinine three grains, may be given night and morning. Maclean considers scruple doses of ipecacuanha, repeated every few hours till the symptoms subside, almost as valuable as in dysentery. Whatever treatment is adopted, rest in the recumbent posture should be insisted upon, and no solid food or stimulants should be allowed. As regards either local counter-irritation or emollient cataplasms, I do not think much benefit, if any, results from their use, but would generally advise a mustard leaf, or soap and opium liniment, more with the view of satisfying the patient than with expectation of other benefit arising from them. If after the subsidence of the acute symptoms pain or tenderness continue, iodide of potassium with a vegetable bitter may be given, and iodine paint may be applied externally. Further treatment will consist in keeping the bowels free by some mineral water (*vide* note p. 438).

HEPATIC ABSCESS.—Hepatic abscess has been classed as idiopathic, traumatic, and metastatic. It may originate, first, suddenly during the progress of either acute or chronic

inflammation; second, gradually after inflammation; third, insidiously, without previously recognisable inflammation; fourth, during the progress of dysentery; fifth, from pyæmia.

1. Whenever in the course of either chronic or acute inflammation or congestion, rigors or cold sweats supervene without any ostensible cause, the probability of abscess is great. But rigors occurring during even acute hepatitis is not an infallible sign, as gall-stone, inspissated bile, or entozoa impacted in the duct may excite shivering. In abscess occurring during the progress of acute hepatitis the diagnosis will be confirmed if rigors are followed by a subsidence of acute pain, by shoulder pain ceasing or becoming intermittent, by short dry cough, tongue red anteriorly, furred posteriorly, scanty and high-coloured urine depositing lithates, cold sweats, hectic fever, emaciation, colliquative diarrhœa, and evidence of enlargement. The latter sign, however, may not present. When the abscess is small and deep-seated, palpation will probably furnish no evidence. The more superficial it may be, the more decided will be the manifestations of its presence. In abscess occurring during acute hepatitis the thermometer remaining high is perhaps the most reliable sign, and this with other general constitutional symptoms is sufficiently demonstrative. Much is to be gathered from the state of the countenance and skin, which are often faithful indications of the progress of the disease. At the invasion of rigors the countenance is pale, and the integument shrunken, and as the suppurative action advances the face becomes fuller in appearance than natural, with some degree of dusky redness in the cheeks, which from the accompanying oppressed breathing must be attributed to imperfect oxygenation. Afterwards the countenance becomes muddy and sallow, with dark appearance under the eyes.

2. A more frequent manner in which abscess manifests itself is after the prominent symptoms of acute hepatitis

have been relieved. The patient does not recover health, remains weak and languid, and after a variable time complains of occasional chills and febrile excitement towards evening, the palms of the hands being unnaturally dry and hot. This may assume a decidedly hectic character, and is accompanied by a tongue furred in the centre but red at the top and edges. Often weight and uneasiness are experienced in the right hypochondrium, with occasionally shooting pains. The appetite is variable and the countenance sallow; the motions probably contain the usual amount of bile, and the urine deposits lithates. There may or may not be evidence of enlargement of the liver, and probably when enlargement does present there may be a temporary accession of pain. Here, again, the general symptoms are often the only guides. When abscess arises gradually after chronic inflammation the symptoms are much as described above, but they are more gradual and obscure.

3. But there is a deep-seated suppuration of the liver which runs its insidious course without any urgent symptoms, without previous fever, inflammation or congestion, sometimes, in fact, without any recognisable symptoms at all. At other times there is a vague sense of uneasiness or obtuse dull pain, or a slight sense of weight or oppression, more or less apparent, according as the disease is centred in the interior or located towards the exterior of the organ. At other times the only symptoms may be indefinite liver uneasiness, a chill or two, and occasional febricula. Such anomalous feelings, with perhaps dry cough and loss of flesh, are symptoms scarcely appreciable by the too frequently doomed patient, or, if observed, are considered too trivial to induce application for medical advice. Sometimes the gradual loss of flesh causes anxiety, but often it is not till rigors occur, followed by cold sweats and tumours, that the serious nature of the disease is understood. Budd, Martin, Peet, Fayrer, and many other authors give instances of this nature.

Dickson states, 'I have known persons die of abscess of the liver who never in life had experienced any of the symptoms.' Copland remarked, 'A person after being resident in India cannot be sure if an abscess exists in his liver or not.' Macpherson has known an iron-worker burst an abscess into his lung when at work, the presence of which was not suspected. I recollect an officer walking into my office (the Deputy Surgeon-General's) in Bombay with abscess of the liver, which he had regarded as a little dyspeptic derangement, and for which he had not sought advice. In fact, the liver may be full of abscess and the person unconscious of it. The idea that abscess of the liver must be accompanied by recognisable inflammation and fever is erroneous. Fayrer stated he had long ceased to believe that rigors and pyrexia were necessary to the development of every abscess of the liver. In these obscure, or, as they may be termed, *latent*, abscesses there is, however, usually a general falling off in health, but so undefined and gradual that it may be attributed, in the absence of all symptoms referable to the liver, to any other cause, and has been mistaken for phthisis. I have reason to believe a person may carry a small abscess in the liver for years, the only manifestation of its presence being slight dyspeptic symptoms, occasional uneasiness in the right hypochondrium, sometimes a recurring or even fixed pain in one spot, and occasional rigors, when apparently the abscess takes on a temporarily active condition, and is probably increased in size. This is more likely to occur in Europeans returned from India.

4. Abscess of the liver occurring *during dysentery* is considered under the heading of that disease (p. 198).

5. Pyæmic Abscess may occur, from the size of a pea to that of an orange. There may or may not be enlargement. Numerous abscesses have been found without enlargement during life. On the other hand, a single abscess superficially situated may cause enlargement, but, as a rule, pyæmic

abscess is multiple. These abscesses commence in death of the cellular tissue, the result of capillary embolism, some containing pus, and some sanious matter and *débris* of tissue. May supervene on dysentery, ulceration of the stomach or of the gall-bladder, injuries of the head, surgical operations, or any cause of pyæmia. Rigors, pyrexia, profuse sweating, and emaciation are symptomatic.

Diagnosis.—Speaking generally, it may be stated that in all cases of hepatic abscess the symptoms will be more or less clearly presented according to, *first*, the size, and, *secondly*, the situation of the abscess, and in many cases they must be considered collectively. Although shivering is often a premonitory symptom, it does not always occur, or may occur so slightly as scarcely to be noticed, while the throbbing which usually accompanies the formation of pus is rarely present. The biliary secretion, which one might expect to be disturbed, is often normal, but white stools may generally be looked on with suspicion when occurring in connection with other symptoms. Bile-pigment is sometimes present in the urine, sometimes not. If the abscess be small and central or posterior, the pressure is so slight anteriorly that the stomach is not affected, neither is pain felt in the side. In such persons the slight accompanying malaise and loss of appetite are apt to be attributed to indigestion. In many cases there is no well-defined class of symptoms which distinctly point out or imply unquestionably that matter has formed. Rigidity of the right rectus muscle may be found when the malady is situated in that part of the liver immediately beneath. Shoulder pain, as in hepatitis, may or may not be present, and is usually felt most when the abscess is posterior. A painful spot at the spinous process of the fourth dorsal vertebra has been sometimes noticed. When the abscess is large the patient is usually chilly, from greater interference with the carbonifying function of the liver. Pointing is not a symptom of much

value, especially in the early stages. It is, indeed, hardly to be expected except in superficial abscess, for by the time a deep-seated abscess reaches the surface it has usually involved a large extent of liver-tissue, and does not exert pressure in a limited spot. The swelling is then uniform and diffused, with often an absence of anything resembling pointing. Fulness and hardness about the epigastrium indicate disease in the left lobe, while cough, impaired movement of the right ribs, shortness of breath, fulness and dulness are characteristic of an affected right lobe. Abscess extending in the latter direction frequently excites pleuritic or pneumonic inflammation, and may be confounded with the results of such diseases as hydrothorax, empyema, or consolidated lung. The history of the case is, however, generally sufficient for diagnosis, and it may be recollected that pain so occurring about the lower part of the right lobe is, in Europe, more frequently due to inflammation of the pleura than the liver, while similar signs in India are mostly indicative of the liver being affected. A friction-sound sometimes exists over a small portion of the area of the liver at the site of the abscess, and this, in one case at least (Sir A. Clarke), has been regarded as sufficiently diagnostic, when coupled with other ill-defined symptoms, to warrant a line of treatment.

A distended gall-bladder has sometimes been mistaken for an abscess, but the pyriform and localised swelling of the former is characteristic. Hydatids and cancer have also been mistaken, and the diagnosis is sufficiently difficult, especially as regards hydatids; for the latter, like some kinds of abscess, cause little constitutional disturbance. The feeling of vibration, often elicited by tapping with the finger over hydatid, cannot be felt in an abscess, owing to the greater thickness of its contents. It should, however, be recollected that in India the probability of abscess is much greater than the probability of hydatid. Still, in some cases the

diagnosis is impossible. I have in recollection the case of a European gentleman, where the disease was only discovered to be hydatid after the patient had been operated on for abscess. As regards cancer, the nodulated protuberance is different from the rounded, smooth, or diffused swelling of abscess. Moreover, cancer seldom occurs before forty years of age, and there is probably a history of heredity. In cases where there is no external budging the diagnosis is still more difficult; as before mentioned, liver abscess has been mistaken for phthisis in its earlier stage, and this is especially liable when there is really phthisis present. The emaciation, hacking cough, evening febrile manifestations, and debility of latent hepatic abscess, combined with flattening beneath the clavicles, vocal resonance, and perhaps a consumptive hereditary taint, is very likely to cause error of diagnosis. The cough of liver is, however, generally dry.

The diagnosis of liver-abscess being so difficult, various authors, and among the more recent Dr. W. S. Palmer, have recommended in doubtful cases exploration of the liver with a long trocar and canula. When pus was not found, it is stated no bad consequences followed. When detected, the puncture caused slight inflammatory action, which ended in adhesion of the organ to the parietes, and so facilitated future opening of the abscess.

Terminations of Hepatic Abscess.—Abscess of the liver may burst into the stomach and be emptied by vomiting. Many instances of recovery are recorded after this termination. It is to be diagnosed by pus being vomited in larger or smaller quantities. 2. The contents may be conveyed along the hepatic duct into the intestines. 3. An abscess may attach itself to, and be discharged into some part of the intestinal tube, as the duodenum, the small intestines, and more frequently the colon, when the pus is evacuated per anum. Sometimes the patient may recognise the period of the bursting of the abscess, and is sensible of the passing of

pus, or pus may be discovered in the motions. This is more likely to be the case if the abscess opens so low down in the intestinal tube as the colon. As a general rule the termination of opening into the intestines can only be suspected by the occurrence of diarrhœa and the subsidence of the liver tumour if any, accompanied by gradual improvement. It is believed that the greatest number of recoveries result from those cases where the pus is discharged either along the hepatic duct or directly into the bowels, and also that there are a larger number of such cases than is generally supposed. 4. An abscess may open through the lungs, and the contents may be discharged by a bronchial tube, of which cough and chocolate-coloured expectoration occasionally streaked with blood or bile are characteristic. In some cases the abscess communicates with a large ragged cavity at the base of the lungs formed by the softening and expulsion of tissue, in which case a cavernous sound will be heard under the stethoscope. Many cases of recovery have occurred after discharge through the lungs, although the prognosis is less favourable than when the pus is discharged into the bowels. Care is necessary lest the condition be confused with the asthenic pneumonia of natives, which is sometimes attended with muco-puriform brick-red sputa. 5. The abscess may discharge into the cavity of the chest and form empyema, which is usually fatal. Under such course of the abscess the history of the case is generally sufficient for diagnosis. 6. An opening may take place into the pericardium, occasioning sudden death by interfering with the action of the heart. 7. Into the peritoneum, producing acute local pain quickly followed by collapse. 8. Abscesses have been known to open into the vena cava and pelvis of the right kidney, of which there are no characteristic symptoms. 9. It may point and open through the intercostal muscles between the right ribs, which are often found necrosed internally. 10. Through the abdominal muscles either beside the ensiform cartilage, at

the epigastrium, or even as low as the umbilicus, known by the usual symptoms of the formation and pointing of an abscess. 11. Pointing may not occur either internally or externally, the walls of the abscess may remain entire, and the patient sink from exhaustion. 12. There is reason to believe an abscess may become encysted and remain latent for an indefinite period, as sometimes masses of cheesy matter enclosed in a thick (occasionally) cretaceous cyst have been found in the liver. Such partially absorbed abscesses are a source of danger, probably giving rise to occasional shivering, to anomalous sensations in the side, interfering with the action of the organ, and are liable to take on active inflammatory action at any time from slight causes, as exposure to chill. It is believed that numbers of persons are thus affected, some who have suffered from symptoms indicating hepatic abscess, and some who have never felt, or at least recognised any such symptoms. 13. There is also strong presumption that liver abscess is occasionally absorbed, as sometimes a white fibrous sac only has been found. Now and then, after unequivocal signs of liver abscess, the swelling and other symptoms gradually disappear without evidence of discharge in any of the ways previously mentioned, and the patient recovers his health. In many of such cases, of which there are undoubtedly numbers, it is probable that although lymph had been effused pus had not really formed. *Lastly*, gangrene has been remarked upon by several writers as one of the terminations of abscess of the liver, but it would seem that cases of so-called gangrene have been merely that black congested and softened state of the organ depending on intense congestion or inflammation, sometimes apparently resulting from the irritation of a trocar.

Post-mortem appearances.—Acute inflammation commences with patches of turgescence and softening of texture, which may vary from the size of a pea to an orange. Lymph may be effused, which may be absorbed or organised into

delicate areolar tissue which does not seriously injure the structure of the organ. In other instances the lymph is converted into pus. Several of these small formations may coalesce, forming one large collection. The walls of the abscess are formed of hepatic structure, which has a ragged irregular appearance, but often the outermost parts of the effusion become organised into a form of fibrous tissue, and a cyst is formed. In recent abscesses and in asthenic subjects no cysts are found. It is believed that when formed the functions of the lining membrane are not confined to the maintaining and isolating the purulent matters, but that it participates in vitality and carries on absorption, which may explain the occasional apparent disappearance of abscess as referred to at p. 428. It is not uncommon to find collections of pus between the peritoneum and diaphragm, or between the latter and pleura, also local peritonitis. The amount of pus found in or discharged from an abscess is often enormous. In one instance an abscess was found large enough to contain a gallon. In another case 600 ounces of pus were discharged in five months. The colour of the pus is sometimes light and healthy-looking, sometimes of a chocolate hue with pinkish streaks, or it may be streaked with bile.

Locality, classes, age, temperament, most favourable to abscess.—Liver abscess is stated to be more frequent in Madras than in either Bengal or Bombay, but this is questionable, as with hepatitis. The greater number of liver abscesses occur in persons not long in the country, especially among young soldiers, but no length of residence confers exemption. As regards *age*, the majority of cases occur in persons in the prime of life, but this probably results from the greater exposure of the classes to the causes of various maladies. No age, however, affords exemption, for a case was recently reported in a boy of seven who had suffered from fever, another in one of twelve, and a third in a child of seventeen months old.

Gordon states there is a certain combination of characteristics which lead a person experienced in the phenomena of disease to suspect a greater liability in those of dark muddy complexion, in the habitually constipated, the dull morose disposition, or in that constitution usually regarded as the bilious or nervous temperament. This, however, is questionable (*vide* remarks on temperament, p. 24), and Mackinnon long since remarked on the greater liability of those of sanguine temperament and lax fibre to hepatic abscess.

The *causes* of hepatic abscess are those of liver disease generally, and of congestion and hepatitis particularly. Harley says emphatically that the chief causes of liver disease in India are 'gluttony and intemperance,' or 'the habitual over-indulgence in rich food and strong drinks,' so that it is 'impossible that all the hydrocarbons admitted to the circulation can be used up,' which, coupled with 'the inactive mode of life followed by the majority of English residents,' produce the liver diseases of India. And Harley is 'led to this conclusion chiefly by the fact that natives of hot climates, whose mode of life is entirely different from that of Europeans, are not one wit more liable to be affected with abscess of the liver than any man residing in Great Britain.'

While fully admitting the noxious influence of too much eating and drinking, coupled with a sedentary life, I entirely disagree with Harley that such habits are the causes, or even the principal causes, of hepatic abscess, and I further challenge the statement as to the gluttony, intemperance, and inactive mode of life of Europeans in India, which is indeed generally characterised by habits the reverse of those imputed to Anglo-Indians. I also say that the statement regarding the immunity of natives is incorrect, as the records of any large native hospital will prove. Still the evil influence of spirit-drinking cannot be denied. Cayley states he has never seen a case of abscess in which the patient has

not been in the habit of consuming alcohol. Gordon also refers liver abscess to the consumption of large quantities of intoxicating liquors. In fact, the increasing frequency of liver abscess among natives has been with good reason attributed to the increased consumption of alcoholic liquors. Macnamara long since showed the mode of life of soldiers was conducive to hepatic abscess and fatty liver. Still numerous cases of abscess occur in very temperate individuals, and in those whose mode of life is unexceptionable in all respects. Thus tropical anæmia undoubtedly tends to induce liver-abscess when hepatic embolism possibly occurs. There is also reason to believe the disease sometimes follows the suppression of hæmorrhoidal discharge. Its connection with dysentery has been already noticed. It would appear, indeed, as if there were some special peculiarity in the climate of the East promoting liver abscess. For among soldiers whose ages, habits of life, and occupation are the same in the East as in the West Indies, more than four times the number of those serving in the East suffer from severe forms of liver complaint. The greater prevalence of hepatic disease in the East than in the West Indies—both hot tropical climates—has never been satisfactorily accounted for, leading Parkes to infer that 'Perhaps after all there is little immediate connection between heat and liver disease;' and others to the opinion that some peculiar climatic influences must exist, a third class falling back on the universal Moloch—malaria. I believe it is simply an effect of atmospheric vicissitudes, nowhere so powerfully felt as in India, where on the coasts the diurnal sea-breezes succeed to a hot moist stagnant atmosphere, where throughout the whole country the night temperature, or rather the morning temperature, is so much less than that of the day, and where in many districts the seasons are so different that they are ordinarily spoken of as the hot and cold weather, while the cutaneous surface of Europeans especially is rendered extra-

ordinarily susceptible to chill by the continued heat. I am supported in this view by the fact, so frequently noticed, of liver disease commencing in the cold season.

In addition to the causes already referred to, hepatic abscess is the occasional apparent result of operation or injury about the rectum; there is also reason to believe it is sometimes originated by a guinea-worm; inspissated bile, biliary concretions, gall-stones forming in the substance of the liver, sometimes excite abscess; it may be the result of pyæmia from various sources; it may be originated during the congestive stage of paroxysmal fevers. Lumbrici have also been found in the centre of an abscess. Syphilitic grummatous deposits may also develope into abscess. Harley refers abscess to localised germs. But I hold the principal causes are chills from multitudinous conditions, and acting with especial force when the organ is predisposed by the congestions, deteriorations, irritations, and structural alterations to which it is so subject in India from varying agencies.

Treatment.—In the first instance, when from the occurrence of rigors or other signs hepatic abscess is feared, endeavours may be made to arrest its progress by the use of cold applications over the part till the skin is deadened, together with low diet and, as recommended by Harley, germicides, especially salicylic acid, may be used. In other respects, much discussion has taken place regarding the best method of treating hepatic abscess, many authorities formerly believing the proper treatment to be supporting the strength of the patient by tonics and nutritious diets, and should the abscess point externally, favouring its discharge by poulticing, fomenting, and position. The integument was allowed to become reddened, and show positive evidence of pointing, or at least of pus, before matter was artificially evacuated; some even advised the skin to be allowed to ulcerate naturally, for, as Dr. Budd pointed out, the solid tissue of the liver cannot readily contract so as to close the

cavity after the matter has been evacuated by a free opening, and all the disadvantages of the entrance of air result. It was argued that the operation of nature so far differs from that of the surgeon that the abscess is never evacuated at once, but gradually, and with much less shock to the system, by one or more small apertures through which the pus slowly drains away. As this aperture is rarely direct, and as the cavity contracts as the matter discharges, little if any atmospheric air is admitted, as is proved by the often observed fact that the discharge in such cases remains free from any offensive smell to the last, while the reverse obtains where an opening is artificially made. Then under the expectant method there is the chance of the abscess being discharged by the lung or bowel, the ultimate result of these natural means of exit being more encouraging than when a direct opening is made. Also the abscess may be multiple, in which case an opening into one formation would have no effect on another. Others, on the contrary, asserted that when we have just grounds for believing abscess exists we ought not to lose a day in evacuating it by puncture, and that we are both justified and safe in endeavouring to hit upon it with a trocar when deep-seated, avoiding the gall-bladder and large veins. This, it was asserted, is less dangerous than allowing the abscess to go on hollowing the liver or endeavouring to work its way into the lungs or bowels, the patient being liable to destruction at any moment from rupture into the pericardium, pleura, peritoneal sac, &c.

Under this idea in former days, especially by Murray, Cameron, Halket, Templeton, Mouatt and others, explorations of the liver in search of abscess were practised in a somewhat coarse manner. Seeing that Indian hakims resorted to puncture of the liver and spleen for the dispersion of indurations and enlargements, Europeans adopted the practice, and repeatedly plunged trocars deep into enlarged livers without finding an abscess, and 'never had the slightest ill-

effect.' Harley also says puncture does not excite irritation; a result which has not accorded with the experience of others, who have reported the reverse (Maclean). The advocates of exploration also stated that punctures have been followed by gradual absorption and disappearance of enlargement, and Webb and others gave cases of the kind. In men who have died from abscess which could not be successfully explored, Cameron found it extremely difficult to detect the mark of the puncture, so that he believed the danger said to attend the operation was argued from analogy rather than practical experience. But granting the dangers to exist, the question presents whether they are greater than those arising from non-interference. Many die without any pointing of the abscess, and the liver is found converted into a mere pus-sac, and the ribs probably necrosed, while the patient is worn out by fever and diarrhœa. It was argued that early puncture stopped this vicious progress, which was to be effected at any place where the slightest œdema or fulness, or pain in breathing, gave an indication, while yet the patient retained some vigour.

The use of the modern aspirator has, however, done much towards removing the objections to both lines of practice. Those who opposed the so-called explorations of the liver in the coarse manner practised in former years, have not the same objections to the use of the fine perforated needles urnished with the aspirator, from the use of which it is difficult to imagine any possible harm. As soon as constitutional symptoms, *e.g.* rigors, night sweats, and persisting high temperature, indicate the formation of pus, the exhausting syringe can be applied, and the abscess explored without pain and without the admission of air. Every authority agrees that temporary, if not permanent, amelioration of symptoms follows the removal of pus from any hepatic abscess. It is, however, often necessary to use the trocar, the aspirator being too fine an instrument for the removal

of masses of thick shreddy pus, which is liable to block. A good deal has been written about the time and method of opening an abscess. Palmer truly states the time to open is when symptoms give an assurance of pus, and the operation necessary to reach it is not so dangerous as to be prohibitive. It may, in short, be stated that opening should be resorted to whenever there is reasonable certainty of finding the pus, as it is desirable to anticipate the opening by some other channels nature may select, and which may be fatal. The aspirator and everything which will help to prevent introduction of air should be taken advantage of, and the precautions of the antiseptic method should be strictly observed. Every precaution should be used to prevent the entrance of air, and the needle should be passed through a pledget of carbolised cotton pressed closely around at the point of entrance, and retained there on withdrawal. The abscess may evidence itself in an intercostal space below the edge of the ribs, at the epigastrium, or as low down as the umbilicus, and it is usual to make an opening wherever there is distinct bulging; but it is better, if possible, to operate below the ribs, as experience has shown the chances of following inflammation or gangrene are less. The relief to pain, tension, fever and general discomfort afforded by evacuating the contents of a large abscess is always marked, and the process should be repeated after a few days.

The rules for operating as given by Fayrer may be summed up as follows: When there is distinct pointing and the abscess is small, an opening may be made with a bistoury at once. When there are no local signs of abscess, but severe and suggestive constitutional symptoms, one or two exploratory punctures with the aspirator will be advisable. When constitutional symptoms of abscess exist, with bulging of the ribs or uniform enlargement, the aspirator should be first used. When in either of the above cases the presence of pus has been confirmed by exploration, it should be aspirated with a large canula under antiseptic precautions. As a general rule, it is best to make a free opening and give exit and free drain to the pus, for which purpose a drainage-tube may be used. But when the abscess is very large it will be better to

evacuate by instalments at short intervals, excluding the air on each occasion. Care must be taken when enlarging an opening, lest liver tissue being cut, hæmorrhage may be excessive.

After opening an abscess a good deal may be gleaned from the character of the pus. If on successive tappings it decreases in quantity and becomes thin and serous, pain and fever subsiding, a cure may be expected. The ultimate adoption of the open method by a free opening or thorough drainage will often expedite the contraction of a large cavity when a decrease in the quantity of pus aspirated indicates that the process has already commenced.

In cases where hope of cure cannot be entertained, *partial* emptying of the cavity at intervals by the close method by means of an aspirator is the wise course to pursue. By this means suffering is relieved, septic poisoning avoided, and risk of sudden death by internal rupture lessened. Completely emptying the cavity is to be avoided, as it favours more rapid refilling of the cavity with broken-down liver tissue and pus.

If after aspiration, gaseous distension of the cavity with constitutional symptoms should occur, it will be necessary to adopt the open method by incision or drainage-tube, or both, for the entrance of air into a closed cavity is more dangerous than into an open one with free drainage. Now the complete emptying of the sac is important, and if this cannot be otherwise obtained, a counter-opening, if practicable, may be useful. In such cases it is often desirable to throw in a weak antiseptic solution.

The administration of an anæsthetic when making an incision is said to be important, both to avoid unnecessary pain and to diminish the risk of internal rupture from the starting of the patient. But as many persons struggle when taking chloroform, the latter object would probably not be attained.

Abscesses have also been successfully treated by drainage-tube without previous incision, and this plan has been ad-

vocated, especially in the neighbourhood of the pleura. If a drainage-tube is used either at first or at a subsequent stage, a spiral iron tube is best, on account of the tendency of the muscles to contract and compress an ordinary elastic tube. Whatever plan may be adopted, a body bandage is desirable, not only as a means of keeping on, but to restrain muscular action. If a drainage-tube is used, it is important to withdraw it as soon as possible, otherwise it may act as a foreign body. McLeod advises it to be shortened in two or three days, and withdrawn altogether in five or six. Twelve to fifteen days is the usual time required for a small abscess treated antiseptically to heal up, but instances have occurred where the abscess has remained open months.

Notwithstanding all that may be said in favour of any method of treating hepatic abscess, the *success depends not so much on the method of treatment employed as on the size of the abscess and strength of the patient.* If the abscess is large and the person weak, the chances of success are small, and probably evacuation by artificial means will hasten the end. If the abscess is small, and the patient fairly strong, the chances of success under any treatment are comparatively increased, and probably evacuation by artificial means will expedite a favourable termination.

SYPHILITIC DISEASE OF THE LIVER.—Many cases of chronic and obscure affection of the liver among soldiers are due, I believe, to syphilis, but the symptoms are ill-defined. The patient usually complains of sense of weight and uneasiness in the right hypochondrium, and there may be some hepatic tenderness. In some cases the liver is enlarged, in others reduced in size. Firm globular elevations may sometimes be felt, and at others it may be ascertained that the relative proportions of the lobes have been altered. There are always dyspeptic symptoms and ulteriorly œdema of the feet and perhaps ascites. In mild or less advanced cases the capsule is slightly thickened, while the surface of

the liver may be indented. After a prolonged syphilitic inflammatory condition the liver becomes much deformed, and appears to consist of a number of lobes, bounded by deep impressions, the substance on section being seen to be traversed by well-marked bands of tough whitish connective tissue. The deposition of gummy tumours or knotty tumours of the liver may occur alone, but they are most frequently associated with some degree of inflammatory action or interstitial deposit. These gummatæ are usually soft in the centre, firm towards the circumference, and of a yellowish white colour, varying in size from that of a pin's head to that of a walnut. They are composed of granular material in the centre, passing imperceptibly towards the periphery into fibro-nucleated structure, and then into a surrounding fibrous zone.

The slow progress of the disease, no history of cancer, a history of syphilis or some syphilitic lesions, combined with the absence of any evidence of chronic alcoholism, are suggestive, if not distinctive. In cirrhosis from alcohol the dyspeptic symptoms are more severe and dropsical manifestations more prominent, while the indications of alcoholism are marked. It is, however, especially in soldiers, sometimes impossible to exclude the idea of alcoholic origin, and probably both causes, syphilis and alcohol, often do combine in exciting obscure affections of the liver. The treatment of syphilitic disease is that of tertiary syphilis, and this can be carried out irrespective of possibility of alcoholic origin if there is a syphilitic history.

Note.—While these pages are passing through the press, I observe Dr. Harley advises, for hepatitis, 'hepatic phlebotomy,' performed by thrusting in a long trocar, keeping away from the transverse fissure, to avoid injuring large vessels and danger of entrance of air. The advisability of this procedure, unless in cases of suspected abscess, may be doubted.

CHAPTER XXIV.

MOON—DISEASES SUPPOSED TO BE CAUSED BY THE.

THE deleterious influence of the moon has been credited from the earliest times, for we have a passage in one of the Psalms, 'The sun shall not smite thee by day, *nor the moon* by night.' We know from Shakespeare that the noxious influence of the moon was believed in at his time. And in most countries during our own time the same was, or is, an article of belief.

The maladies most credited to the influence of the moon are swollen face, tooth-ache, deafness and ear-ache, rheumatism, paralysis, paralysis of the bladder, dropsical effusions, mental excitement or insanity, blindness, and paroxysmal fever. I have known all the above follow, although I do not say caused, by sleeping or lying in the moonlight. That the rays of the moon exert an injurious effect has received support from the allegation of meat becoming tainted sooner on a moonlight night than at other periods. This, however, does not occur if the meat is sufficiently protected. When meat becomes tainted during a moonlight night it is from the operations of insects, which the moonlight lures from the retreats they pass the dark nights in, and not from the moonlight itself.

As regards swollen faces, tooth-ache, and rheumatism, I have always regarded such conditions, when following sleeping in the moon-rays, as the natural result of exposure to damp from dew or to cold winds, which must be the consequence

of lying without any protection from the rays of the moon; for the absence of protection from the latter must also entail want of shield from the former. Similar remarks apply to deafness, with or without an inflammatory condition of the ear.

As regards paralysis, this is a misnomer. The cases of so-called paralysis are really merely the benumbing of a limb, or of some of the facial muscles, from cold and rheumatism, and it will always be found that although a person after lying in the moonlight may not be able to walk or to raise his arm with facility, still he is able to move the affected limb to some extent. Of course, it might happen that a person might be seized by paralysis when sleeping in the moon-rays; and one such case would go far to establish, in the opinion of the ignorant and credulous, the fact of paralysis being caused by the moon. But ordinarily the so-called paralysis is as above described, and it is quickly recovered from. Moreover, the affection, like all other maladies attributed to the moon, really occurs from exposure on other than moonlight nights. Maladies of the kind were in former days described by the older authors as barbiers (*vide* p. 48).

Paralysis of the bladder, leading to inability to make water, occurs from night chill, and has been erroneously attributed to the influence of the moon. This is more likely to occur in men suffering from stricture or irritable bladder, especially if they have been drinking.

Dropsical effusions sometimes result from exposure, and have been attributed to the moon. Thus sudden acute œdema, and the acute form of beri-beri have been ascribed to the influence of the moon, and not to their real cause—exposure to damp, chill, and cold land winds.

Mental excitement and insanity have, I believe, been developed by the moonlight in persons who are constitutionally disposed to insanity. It is a well-known fact that lunatics are always more excitable and noisy in the brilliant

moonlight of the tropical night than at other periods. This results from several causes, the principal of which is the light *per se*, which tends to prevent sleep. Probably the same effect would be induced by the electric light. Then a tropical moonlight night is more noisy than a dark night. Birds, animals, and insects are more restless. Crows caw, dogs bark, and various insects, which on a dark night are quiescent, often show activity. As mental excitement is thus caused to the confirmed lunatic, so those predisposed to insanity are kept awake and excited by the brilliant moonlight nights of the tropics, and the absence of silence consequent thereon.

Blindness more or less complete, like the former ailment, is sometimes caused by moon-rays, which shining on the face of the sleeper cause a congested and semi-paralysed condition of the retina. Sometimes the ailment simulates night blindness, at others day blindness, but more commonly the blindness is both by day and night.[1] I never knew this occur except after moonlight nights, and in the Persian Gulf I recollect various cases. The affection is most likely to occur in persons tainted with scurvy. Unless there is such a taint recovery is rapid under confinement for a day or two in a darkened apartment; but sometimes the origin of future mischief appears to be laid. This form of blindness is sometimes feigned, but as a general rule the schemer pretends to too complete blindness; for although the malady is conveniently termed blindness, it is rather a dimness or imperfection of vision than blindness. Also a schemer does not recover so rapidly as cases of the real moon-affection.

Of all maladies which have been supposed to be caused by the moon, paroxysmal fever has been most favoured as a moon ailment. The connection of fever with the *phases* of the moon, but not, as with the maladies previously mentioned,

[1] The terms nyctalopia and hemeralopia are not used, as they have been applied differently by various writers.

with the moonlight, is yet credited by many Europeans, especially forest officers, and by the majority of natives. In the article on Elephantiasis (p. 237) several authors are mentioned who connect elephantoid fever with the phases of the moon. Sir Charles Napier wrote from Sind, 'Whatever may be the explanation by doctors, down we all go with fever at the new and full moon.' Morehead attempted an explanation by opining that at the phases of the moon the tides left exposed a larger amount of malarious surface, which with other subtle atmospheric conditions of the period affected the constitutions of those prone to fever. Notwithstanding the almost universal belief of accessions of paroxysmal fever being more frequent at the new and full moon, there were still those who doubted the fact. Years ago, previously to a weekly mail from England, and when the mails did not arrive so regularly as they do now, there was a general impression that the mail steamer nearly always arrived in Bombay harbour on a Sunday. When, however, the arrivals were tabulated this was found not to be the case, and evidently the impression had resulted from the arrival of the mail on a Sunday—when persons had no office work to occupy them—being better recollected than when it arrived on other days. So it was argued attacks of fever were more noticed and better remembered when occurring at such notable periods as the new and full moon are in the East, than when attacks occurred at other times. With the view of settling this question a large number of febrile attacks occurring in the regimental hospitals were tabulated, and it was found there was no increase at the new and the full moon. The registration of a large number of attacks occurring in the hospitals and dispensaries of Rajpootana instituted by my directions,[1] gave precisely similar results. It may therefore be confidently stated that, notwithstanding the still confirmed belief of the masses, there is no

[1] *Ind. Med. Gaz.*, 1867.

connection whatever between the phases of the moon, or between the tides, and paroxysmal fevers.

At p. 9, the objections raised by executive officers in former days to spreading awnings on board ship is referred to. All the maladies previously mentioned were developed by the absence of awnings, not because the awnings intercepted the moon-rays but because they shielded the men lying on deck from damp, dew, and to a great extent from cold winds.

CHAPTER XXV.

NEURALGIA.

It is not so generally remembered as it perhaps should be, that although neuralgia may be purely local as regards the chief symptom, viz., pain, it has very commonly, if not always (excepting when caused mechanically), a constitutional origin. The painful affection we term neuralgia may attack almost any nerve in the system, although there are some nerves more prone to the malady than others. Referring to the different classes of nerves, those lying superficial are more disposed to neuralgic pain than others more deep-seated. Of the superficial nerves the trifacial, the sciatic, and the intercostal, are most frequently affected. In this article it is proposed to refer only to the nerves of the face, and chiefly to that portion of the nerves of the face implicated in Brow-ague.

In addition to the term 'Brow-ague,' or 'Brow-ache,' we find the affection frequently referred to, not only by the public, but even by professional men, and also in systematic treatises in medicine under various different names. Thus some use the expression *Neuralgia faciei* to designate not only brow-ague, but also pain occurring in any other part of the face. Others prefer the term *tic-douloureux* for any form of pain affecting the sensory nerve of the face, whether it is the supra-orbital acted upon by malarious or other constitutional influences, or whether it is the superior or inferior maxillary, irritated by decayed teeth or diseased gums. *Hemicrania* is also used when the pain assumes the

peculiar character of limitation to one side. *Megrim* again has been applied to pain referred to the internal angle of the orbit and side of the nose. *Migraine* is another appellation which, borrowed from the French, is much in vogue with some persons for almost any kind of headache. Lastly, a more purely scientific nomenclature has been attempted, as *cephalalgia muscularis*, *cephalalgia periosteosa*, and *cephalalgia neuralgica*; the first being regarded as rheumatic, the second as syphilitic, and the third as neuralgic or periodic, comprising all the terms above-mentioned, viz., brow-ague, *tic*, *hemicrania*, *megrim*, and *migraine*. In the official 'Nomenclature of Disease' the heading 'Neuralgia' is divided into (*a*) *neuralgia faciei* (facial) with the synonym *tic douloureux*; and (*b*) *neuralgia frontis* (brow-ague) with the synonym *hemicrania*. Perhaps the official nomenclature is the best and most simple which has yet been devised; and it would be well if, when speaking or writing on the subject, one set of terms only were used, as the frequently loose manner in which the various appellations are applied often gives rise to doubt as to what particular form of disease is really the object of comment. *Neurosis* is a word modernly employed to indicate a malady which depends on some perverted nervous influence rather than on merely local change, and this would appear to express the position in the maladies in question more precisely than the term neuralgia, which, derived from *neuros* nerve, and *algos* pain, simply indicates the meaning of nervous pain, or pain of a nerve.

The terms *neuralgia frontis*, or brow-ague, or brow-headache, or *hemicrania*, are generally applied to pain of a very severe character affecting the forehead generally above one eye, and that most frequently the left; but often the pain is more diffused, sometimes over the one side of the head. In typical instances the pain shoots from the spot where the nerve issues through the superciliary

foramen, and may sometimes be sharply traced and defined by the finger or even with the point of a pencil. In a few instances, when this occurs there is a visible red line in the track of the nerve. But in by far the majority of cases, and especially in old-standing or recurring instances of the disease, the pain extends to the adjacent parts, to which the fibrils of the nerves are distributed; not only the forehead and brow, but also the upper lid and sometimes the eyeball itself participating in the pain. In a minority of instances the pain is more or less defined over half the side of the head. Often it has a superficial character as if in the skin, and at other times it has a deeper-seated character as if in the superciliary ridge of the frontal bone. The pain while it lasts is often most intense and increases in paroxysms, with alternate intervals of comparative ease. The accessions of pain frequently cause the eyes to water and the nose to discharge, while the adjacent arteries throb, and the conjunctiva often becomes blood-shot. Severe attacks of the above description utterly unfit the patient for any kind of business; but persons often suffer from a minor degree of the malady, which may be so slight as to scarcely attract notice, or may be simply an inconvenience. The pain may persist during the whole day or during days, but ordinarily attacks of 'brow-ague' subside in a few hours. When there is also dyspepsia, the 'zigzag' halo of light, described at p. 218, may present.

True 'brow-ache' or *neuralgia frontis*, as above described, is always more prevalent in those localities which we are accustomed to regard as 'malarious,' and often is, especially in the first instance, the result of exposure to those influences which we understand by the term 'malarious.' As with other maladies coming under the same denomination, 'brow-ache' is also characterised by periodicity, recurring often at regular intervals, as every or every second day, with the same pertinacity as an intermittent fever. In a few

instances it may be found alternating with a paroxysm of the latter disease, and sometimes it has appeared to occupy the place of the cold, or of the cold and hot stage, disappearing on the formation of cutaneous exudation. When brow-ague has thus occurred in connection with intermittent, there may be a tendency to the former affection long after the latter has disappeared.

But although 'brow-ague' may be, and often is, caused by what is understood as malarious influences, a very similar pain may arise, and perhaps more frequently does arise, as the result of dyspepsia, or of stomach, or bowel, or liver derangements of a functional nature, of the existence of which the brow-ache may be almost the only and certainly the most prominent sign. There is reason to believe that numerous cases of brow-ague or brow-ache have been treated as malarious with no good effect, when remedies directed to the organs named, with care in diet, would have been followed by immediate success. But in India especially we are too prone to refer every malady, characterised in the least by periodicity, to malarious influences, and to treat with so-called anti-periodic medicines. Yet the fact is, that most if not all diseases are more or less characterised by periodicity, whether their cause may be so-called malaria or anything else. When a person is seen suffering from brow-ague without any very evident symptoms of stomach, liver, or bowel derangements, without any of the symptoms of dyspepsia or indigestion, with a clean tongue, with bowels acting regularly and naturally, and with every function according to the patient's account in excellent working order, it may be difficult at first to appreciate the fact that a functional error so slight as to be thus overlooked can be the cause of frontal pain. But an attentive examination will seldom fail in discovering something wrong of which the patient has no idea, or which he considers of no consequence. Perhaps the patient may be passing oxalate of

lime, or albumen, or sugar in his urine, with the loss of all of which frontal pain is sometimes associated; just as it may be with menorrhagic or with hæmorrhoidal discharges. There may be, unknown to the sufferer, white stools, showing the existence of at least some hepatic functional derangement. There may be either diarrhœa or constipation to an abnormal extent, but which the patient in his ignorance regards as natural. There may be acidity of stomach, and errors of diet which the person considers too trivial to mention. There may be torpidity of, or even accumulation in, the large bowel; a condition more frequent with persons long in India than is generally suspected. Also, especially in children, the existence of vicious habits of more than one description, which by their drain upon the system produce nervous debility, and its almost universal accompaniment, nervous pain. There is, however, nothing conclusive in the character of the pain to establish a diagnosis between that arising from so-called malarious influences or that arising from other causes. But as some guide, it may be observed that first attacks especially of malarious brow-ague are more frequently sharply defined, and more localised, than brow-ache from other causes, in which, as a rule, the pain is more diffused over the side of the head. But after repeated attacks from malarious influences the pain is not so well defined as it is generally at first.

In addition to malarious influences, and to dyspeptic derangements, 'brow-ache' may arise from almost any cause which lowers the vitality of the system. Thus when women have been subject to weakening agencies, such as frequent child-bearing, prolonged suckling, or profuse menstruation, they are additional causes favouring the occurrence of the malady; so much the more certainly if the person has been residing in malarious, hot, and therefore debilitating, climates. The effect of repeated and continued, although small, loss of blood, from any cause, in weakening the ner-

vous system, is well understood. The result of continued heat is none the less certain. In addition to the acknowledged *direct* depressing effect of heat on the nervous system, there is the degenerated, or it may be said the poisoned condition of blood arising from a heated and therefore rarified and comparatively less oxygenated atmosphere, combined with often too little exercise, and generally too much food. This also tends to a debilitated condition of system and a proneness to nervous complaints. There may be in addition overwork in badly ventilated apartments ; and especially overwork of the brain, which is a fertile source of nervous pain in one or other position about the head.

Thus brow-ague or brow-ache may depend, and *most frequently does depend*, on a combination of the causes previously referred to, partly on one cause and partly on another. It may be due to what is understood as malarious influences, and may be aggravated or re-excited by stomach or liver derangements, or by a low condition of the vital powers ; or it may be due to stomach or liver derangements, or dyspepsia, or a low vitality, and be aggravated or re-excited by malarious influences. This, the most common variety of the disease, is most frequently met with in sensitive and nervous people, especially after mental worry or excitement of any kind. Any cause in fact which produces an impression on either the nervous or digestive system of those disposed to the malady will bring on an attack. Food to which the person has been unaccustomed, even although taken in minute quantities, exposure to heat or cold, damp, working at night, anger, excitement of any kind, will cause a paroxysm. In women who have suffered from the disease it frequently returns at or about the monthly periods. Those thus predisposed, often without any very evident cause, may wake in the morning feeling sick, with the temples throbbing, with hot palms, and more or less unable to eat, move, or converse. The head feels hot, and the application of cold is generally

refreshing. But the patient most frequently prefers being left alone, and finds quiet the best mode of obtaining relief.

Brow-ache, or indeed neuralgia in general, has been attributed to a 'morbid state of the nerves of sensation,' to 'defective nervous energy,' to 'deficient vitality,' to 'nervous exhaustion,' to 'lowered nerve force,' to 'nervous irritation,' all phrases leaving us as much in the dark as before respecting the true causation of the disease. Attributing the malady to 'malarious influences,' to over-work, to dyspepsia, as in the preceding remarks, is, it must be confessed, scarcely more satisfactory unless it can be shown in what manner these excitants operate. Yet, in determining the cause of the malady, there is but little aid to be found in conditions which in some other diseases are of the greatest assistance. For instance, pathology gives us little or no evidence. Nerves affected by neuralgia have been carefully examined after death, and have been found, to all appearance, healthy. It is true that in some few instances redness or atrophy of a nerve has been described, and there is reason to think that in a great minority of cases inflammation of the neurilemma may be a reason for neuralgic pain. But this certainly is not the case in by far the majority of cases. And when redness or other changes of the nerve have been found, they are not more than what may be expected, and are the *consequences*, rather than the *causes*, of the existence of the disease.

As there is nothing to be gained with reference to the cause from the pathology of the malady, other circumstances, as the class of persons in whom it occurs, the condition under which it occurs, and the localities in which it occurs, may be inquired into with the view of throwing some light on the causation of the malady. As before observed, the dyspeptic, and the debilitated from any reason, are the most likely to suffer from brow-ache. In England it has been noted, that neuralgic complaints are comparatively more

numerous among the poor and labourious classes—a remark which would be equally applicable to chronic rheumatism. Moreover, it has further been observed that the disease is still more prevalent in persons who are not only dyspeptic but who have also been exposed to some debilitating influence, or to what may be understood as 'malarious influences.' As regards the conditions under which it occurs, there is nothing more likely to be followed by an attack than exposure to extremes of heat and cold, or in a hot climate exposure to a very minor degree of cold—such, for instance, as the exposure to the night air in an open carriage or from sleeping in a draught. The next condition under which it most frequently happens is that of minor dyspeptic derangement or excitement, from often a very slight departure from accustomed hours and habits, and this the more certainly if the patient has been also subjected to mental work or mental worry. As regards the localities in which it is most prone to present, these may be in Europe mentioned as low, damp, and malarious. But in India hill-stations seem to be perhaps more conducive to brow-ache than other localities. This has been remarked by myself at Aboo, and by others at Nynee Tal, and at least one other of the Himalayan stations. The explanation of this may perhaps be exposure to a greater degree of cold by persons who have long been habituated to heat, and who are in some way or other rendered prone to the malady from dyspeptic or debilitating conditions.

It is well known that nervous pain may be excited by some remote irritation. Examples of this sympathetic neuralgia are pain in the shoulder during affections of the liver, pain in the *glans penis* from stone, pain in the testicle from irritation of the kidney, pain in the arm from disease of the heart, pain in some part supplied by the trifacial from a diseased tooth. Pain may also result from mechanical irritation or from pressure, as from the lodgment of a foreign

body in some part of the course of a nerve, or from the growth of a tumour or bony exostosis in such position. The last class of cases forming, so far as facial neuralgia is concerned, but a very small number, may be excluded from the consideration of the causation of the malady as it occurs to the great majority.

Remote nervous irritation is, I believe, the origin of brow-ague and, in by far the greater number of instances, this irritation is the direct consequence of some abdominal or stomach derangement. I do not believe in any such thing as a local neuralgia unless caused by a mechanical irritation. That local disease causes constitutional suffering is not an observation of recent times; but it appears to have been often forgotten that local disease must first be caused by some constitutional error before it could again react on the constitution. For instance, disease of the knee-joint may be, and indeed often is, the first indication of activity of scrofula in the system, and the local disease which the scrofula excites again reacts on the system. Similarly, tertiary sores may be the first indication of the syphilitic poison remaining in the system, the syphilitic sores soon reacting on and further debilitating the sufferer. Similarly, nervous pain, especially brow-ache, may be the first indication of dyspeptic derangements, the pain after a time itself reacting on the system, so that it (the pain) may be accepted as the disease instead of what it should be regarded as, viz., the sign or symptom of a disease—which fortunately is often functional. If it is asked *how* stomach or liver derangements affect the distant nerves, and *why* they affect the supra-orbital nerve in particular, the reply must be, that we do not know; and this answer will be quite as satisfactory as referring to 'reflex action' or to lowered nerve force in explanation. However this may be, the practical fact remains, that brow-ague is most generally, if not always, connected with dyspeptic conditions, of which the following

observations afford proof. Even severe forms of facial neuralgia are often immediately and effectually relieved by sal volatile, by carbonate of soda, by seltzer water, or even by a draught of plain cold water. Most authors on the subject moreover mention the great benefit to be achieved by medicines directed to the improvement of the action of the stomach, liver, and bowels. Every author also acknowledges the failure of the numerous local remedies which have been recommended. Independent of some mechanical irritation, in which category may be included morbid growths, and independently of diseased teeth or gums, it is believed that the *fons et origo* of brow-ague, and indeed of facial neuralgia generally, is to be sought for primarily in dyspeptic derangements, and secondarily in anything tending to debilitate the nervous system. Further, the immediate exciting cause of the pain may often be exposure to cold.

Under such a view, the *treatment* must consist in especial attention to the digestive organs, with the view of ascertaining what may be amiss; not always an easy matter. In cases where there is evident stomachic or hepatic derangement, the plan of treatment will be clear, but in other instances where dyspeptic manifestations are not so strongly, and sometimes very imperfectly marked, the remedies will not be so obvious. But the rule, which in the great majority of cases will be followed by success, is to use purgatives moderately. In plethoric habits, and when the constitution has not materially suffered by protracted agony, the aperient plan should be steadily persevered in, and carried to its full extent by daily doses of the medicine chosen. The diet, which should be carefully regulated, should consist of light and nutritious food, and anything which from experience is known to disagree should be avoided. In such cases also, spirituous and fermented liquors should often be absolutely interdicted. The aperient used may be the good old formula of blue pill and colocynth with an aperient draught a few

hours afterwards, or when the bowels are evidently costive croton oil may be given with extract of colocynth and henbane; or podophyllin may be used when there are symptoms of hepatic derangement. Even when the malady occurs in weak and delicate persons, as for instance in anæmic females in whom the periodical functions of the uterus are disturbed, aperients should still be resorted to, although with more caution and in smaller doses. For such persons, podophyllin, in combination with compound rhubarb pill and henbane, will be found a desirable formula. Although thus laying stress on the necessity of in all cases using aperient medicines, the utility of tonics is not denied or forgotten. Quinine, iron, and the vegetable bitters will all be of service; but as a general rule, only after a course of aperient medicine as above recommended. While a regular periodicity of recurrence will indicate the use of quinine, the anæmic or chlorotic condition in females will point to the desirability of iron. But the routine administration of sesquioxide of iron or other tonics, as sometimes recommended from the first, and without especial reference to the state of the digestive organs, is more likely to be followed by failure than by success. The good results which have frequently followed the administration of larger doses of hydrochlorate of ammonia (chloride of ammonium) are probably due to its action as a laxative and diuretic rather than to any specific effect over the pain. It may be observed (*en passant*) that hydrochlorate of ammonia is more beneficial in pains of a rheumatic character than in the true neuralgic malady. The same may be said of iodide of potassium, which sometimes appears to do good. Colchicum, sulphur, and turpentine have been used with success sometimes; their action on the bowels probably accounting for the recommendations they have received. When the glimmering or halo of light, mentioned at p. 446, occurs the person should take a stimulant and lie down on the opposite side.

But immediate relief from the pain is sought for by the patient, quite as urgently as permanent cure. As before observed, this is sometimes to be obtained by a dose of sal volatile or carbonate of ammonia, or carbonate of soda. If the irritating cause is acidity of the stomach, such use of antacids is frequently followed by immediate relief. If these remedies are not at hand, a draught of cold water or tea will often be beneficial. But if there is reason to suppose the stomach occupied with indigestible food, the use of antacids, as above, will scarcely prove sufficient, and quicker relief will be experienced from an emetic, as twenty grains of ipecacuanha in a cupful of warm water. In first attacks of the disease, which may have come on in connection with ague, or after exposure to so-called malarious influences, after a laxative, quinine may be given every three or four hours. If there are a feeble pulse and fainty feelings, a little wine or brandy and water will be desirable. Sometimes hot, but generally cold applications are the most beneficial. Ice, ether, eau-de-cologne, or hydrochlorate of ammonia and salt, may be employed as cold applications. If heat is required, fomentation with hot water may be used, or a quantity of salt may be heated, placed in a handkerchief, and bound round the forehead. Neither must pressure be forgotten. Shakespeare, who was almost as great a physician as a poet, causes Desdemona to bind Othello's head, when the latter, as the result of watching and fatigue, had pain in his forehead, and which simple remedy is said to cure Othello 'within this hour.' Chloroform applied to the part is often useful. A piece of lint may be saturated with this liquid and applied to the part, and the effect is more decided if the lint is covered with a watch-glass or wine-glass to prevent evaporation. A drachm of camphor, dissolved in half an ounce of spirits of wine, or in sulphuric ether, may be applied with a piece of sponge, or lint fixed to a stick, which will produce a partial loss of sensation. Tincture of opium, tincture of

aconite, and chloroform, in equal parts may also be used. Cocaine solution has been much and successfully used of late. Or a small mustard poultice may be placed on the forehead. A rapidly acting blistering fluid has been recommended, and the after-application to the blistered part of half a grain of morphia. Belladonna is a favourite application with some. Aconitina used as an ointment, in the proportion of two or three grains of the alkaloid to once ounce of lard, has also been extolled. The subcutaneous injection of one-sixth to one-quarter of a grain of morphia, either at the painful part or in some other position, will give temporary relief, and is advised for those patients who cannot afford to lie up. Tincture of gelseminum (the yellow jessamine) is also advised by American practitioners, both as an external application and for internal use, in doses of five or six minims. Its action is said to be analogous to that of aconite. The application of ether by means of the spray is often one of the most certain means of affording relief, although it sometimes fails. Lastly, in some cases, violent bodily exercise has been known to relieve neuralgic pain of the forehead.

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CHAPTER XXVI.

PLAGUE.

Plague has frequently occurred in an epidemic form from the earliest time to the present. For example, in Egypt, in A.D. 361; the 'black death' of the fourteenth century is supposed to have been plague; then there is the great plague of London, A.D. 1665; at Marseilles, 1720; in Western Arabia, 1853; and during most recent years in Persia and Turkish Arabia. It is questionable, however, if true plague has occurred in India, although a disease presenting very similar characteristics has been noticed at various times. In 1827 what is known as the Palli plague, or *mahamuri* (great death), commenced in the city of that name in Marwar, in Western Rajpootana, and spread northwards through the country as far as the Himalayas. In 1859 I saw persons suffering in Kutch from fever of a typhoid type attended with boils on the body, and various similar instances have been recorded.[1] Recollecting the contagious character of plague, that the disease prevails periodically on the banks of the Euphrates, that communication with Western India is frequent and rapid, and that there is a large population in India living under precisely the conditions which favour plague in other tropical and semi-tropical countries, it may be esteemed fortunate that plague has not yet been added to the list of ever-present Indian diseases, which, however, might occur at any time.

[1] *Vide* author's 'Sanitary Progress in India,' *Calcutta Review*, 1876.

Dr. Colville, of the Bombay Service, who was several years surgeon to the Bagdad Political Agency, has given much information with regard to plague. It is described as a specific fever, attended with swelling and suppuration of the inguinal and other glands. The first symptoms are lassitude, shivering, vomiting often of a black material, a heavy stupid expression of countenance, and redness of the conjunctivæ. This is followed by high fever, with darting pains in the groins or armpits, where large boils or buboes quickly form. Sometimes a bubo is almost the first symptom, and so sudden is the advent that the patient feels as if he had been stabbed in the part. There is often, also, petechiæ on the body, sometimes very thickly placed, causing the skin to assume a livid hue, and accounting for the term which has been applied to the disease, 'black death.' Sometimes blisters also form. There is now a special physiognomy, the aspect is haggard without the fixity of typhus, the eyes are retracted but without the blue circle of cholera, and there are no wrinkles as in cerebral maladies. Profuse perspirations, diarrhœa, epistaxis, and delirium occur. The duration of bad cases is only two or three days, but less severe cases may be prolonged as many weeks. Several varieties have been described, as *fulminant*, when death takes place in a few hours with vomiting of blood; *abortive* or *ambulatory*, when there is little fever, and the swellings which occur may or may not end in suppuration.

Causes.—Cabradis styles the disease *miseriæ morbis*, as it is chiefly the poor who suffer, but the well-to-do are not exempt. The conditions under which plague arises are a moist semi-tropical atmosphere, a low-lying alluvial soil near the banks of rivers, crowded and badly ventilated dwellings, emanations from decaying animal or vegetable matters, insufficient and unwholesome food; the combination leading to physical and moral wretchedness and deterioration. When thus originating it may spread to other populations, and to

other places less influenced by the conditions named. The more closely the healthy are brought into communication with the sick, the more certain are the former to suffer. But there is no certain evidence of actual contact exciting the disease, although it seems probable that it may be conveyed by clothing or bedding. The influence of season is marked, more cases occurring in the coldest weather, which, perhaps, may be explained by there being more overcrowding at such times than in the warmer periods of the year. The incubation is believed to be about five or six days.

Treatment consists in affording a pure atmosphere, in giving nourishing food, stimulants and tonics, in isolation and disinfectants, and in treating boils and buboes on established surgical principles.

CHAPTER XXVII.

SCURVY.

Synonym: *Scorbutus.*

Definition.—A constitutional disease traceable in most cases to a definite violation of hygienic requirements, particularly to defective and improper food, manifesting itself by general cachexia and local disorders of a hæmorrhagic nature.

Antiquity and prevalence of scurvy.—Scurvy has prevailed at various times, in every country, in every season, and in every variety of the human race; but unless under exceptional circumstances it is, at least in its minor manifestations, more common in eastern tropical countries than elsewhere. This doubtless arises from the climate and circumstances of life of the residents in such countries, many of whom are forbidden by their religion to consume meat as an article of food; most of whom are previously affected by malarious influences, a considerable proportion being poor, and, therefore, underfed, even when their habitual dietary is taken into consideration, and where fresh vegetable food having anti-scorbutic properties is scarcely procurable by the masses. That scurvy has always prevailed is evident from the description, although somewhat vague, of the malady given by Hippocrates. Scurvy is also mentioned by Pliny as having occurred in the Roman army commanded by Germanicus, after a long encampment in Germany beyond the Rhine. It prevailed in the army of Louis IX., in 1260, when deprived of means of sustenance by the overflow of the Nile. It also

prevailed generally among the Crusaders. That armies in recent times are especially liable to scurvy is sufficiently evident from the history of the Crimean War of 1854; from that of the Indian Mutinies of 1857; and from that of the American War of 1862. For however well the causes and treatment of scurvy may be now understood, and however much it may be desired to counteract the disease, the circumstances of active military service, viz. diet insufficient in quantity or deficient in quality, fatigue, and exposure, are precisely those conditions which render the system liable to scorbutic affections. In fact, the principal rôle of scurvy has been during recent years that of a war pestilence; and holding in view how the scorbutic condition aggravates many other diseases, we see the force of Parkes' observation, that 'If scurvy could be prevented in armies, every other war disease would be comparatively trifling.' Scurvy was so prevalent during the siege of Paris by the Germans that several recent continental authors (viz. Villemen, Rottwil) revived the opinion maintained by some of the older writers of the contagious nature of the disease.

The faulty dietetic and other circumstances in which sailors were formerly placed, resulted in the scurvy being at one time regarded as inseparable from a life at sea, and until late years scurvy carried off from one-sixth to one-tenth of a ship's company during most long voyages. In 1498, Vasco de Gama lost 100 out of 160 of his companions between the Cape of Good Hope and India. In 1726, Admiral Hosier twice lost his crew from scurvy in the West Indies. In the *Centurion*, the ship in which Lord Anson (A.D. 1740) made his memorable voyage round the world, in the latitude of the island of Juan Fernandez, forty-three men died in one month and double that number during the next. In 1795 the safety of Lord Howe's Channel fleet was seriously endangered by scurvy. In 1797 the mortality of the whole of the British naval force was 125 per 1,000. Forty years after-

wards, or in 1879, the death ratio had fallen to thirty-one, principally from the lessened mortality of scurvy. Now, scurvy may be regarded as almost extinct in the Royal Navy, while in the mercantile marine it is less by 80 per cent. than it was previous to the Merchant Shipping Act of 1867, which provided for a supply of antiscorbutics during long voyages. It may indeed be stated, that the freedom of sailors from scurvy in a well-regulated ship at sea is nearly equal to those living in favoured regions ashore. This immunity is attributable to the systematic use of lime-juice, aided by better accommodation, the use of preserved provisions, and shorter voyages under steam. Lind, so far back as 1757, proved the efficacy of fresh vegetables, and especially of oranges and limes; but his recommendations were not adopted by the Admiralty for more than forty years afterwards. This is scarcely matter for surprise, for although it is now well recognised that there are few maladies more easily prevented and more readily cured than uncomplicated scurvy, we have so recently as 1875, in the history of Sir G. Nares' Polar expedition, an example of the accession of the disease consequent on the neglect of necessary precautionary measures.

An account of the ravages of scurvy, in association with want among civil populations, may be found in the history of the wide-spread epidemic occurring in Russia in 1849; in Ireland in 1846; and to a smaller extent in the history of Indian famines.

Anatomical characters and pathology.—The disease produces the effusion of a semi-organised fibrinous material into the tissue of the gums, between the fibres of the muscles, and into the cellular tissue, the lower extremities being ultimately most severely affected. As careful examination of the small blood-vessels and capillaries has shown nothing abnormal excepting occasional very scattered fatty granulations usually found in other maladies, the effusion has

been regarded as due to a faulty state of the blood itself, and not to a degenerated condition of the blood-vessels.

In the endeavours to determine the pathology of scurvy, attention has been principally directed to the condition of the blood and the condition of the urine, and certain depraved states of these fluids have been regarded as exactly those features which might be altered by well-established anti-scorbutic agents. But it would appear that the constituents of the blood and urine differ at various periods of the disease, perhaps consequent on remedies or food taken by the patient. Thus the red corpuscles have been found diminished in proportion, while other observers have considered them normal in quantity. They have also appeared to some presenting a shrivelled or disintegrating aspect; others found no anomaly in form. The blood has also been found rich in globulin and iron; or at least containing these matters in the proper proportion, and the reverse. Fibrine, albumen, and salts have been found increased, normal, and diminished. Colourless corpuscles have been thought to be increased; but this appearance might be due to a smaller number of red corpuscles. Sometimes the blood has been observed to be light and not coagulating firmly, at other times the reverse. In short, the condition of the blood shows no peculiarity which might not occur in other pathological processes.[1] As regards the urine, it has been shown by Ralfe and others that there is usually diminution of potassa in that of scurvy patients, but in the first stages especially there are abundant chlorides.

Dr. Garrod[2] has the credit of suggesting that the cause of scurvy lay in the diminution or withdrawal of some of the alkaline constituents of the blood. So long since as 1848 he made the observation that in scorbutic diets potash existed in smaller quantities than in anti-scorbutic ones, and that

[1] Ziemssen, *Cyclopædia of Medicine*, vol. xvii. p. 177.

[2] 'Nature of Scurvy,' *Edin. Med. Journ.*, vol. ii.

the amount of potash in the blood and urine of a scorbutic patient was diminished. Also that in the typical anti-scorbutic fruits and vegetables (lemons, cress, cabbage, &c.) alkaline bases abound. Parkes was inclined to believe scurvy is caused by a deficiency of the combinations of alkalies with those acids which form carbonates in the system, viz., lactic, citric, acetic, tartaric. Anderson attributes scurvy to a deficient supply of phosphoric acid. Ralfe considers the alteration in the blood in scurvy to depend on a general change between the various acids inorganic and organic, and the bases found in the blood, resulting in diminution of the normal alkalinity of that fluid. Acid salts are constantly entering the system with the food, are being generated in the alimentary canal, and in the tissues of the body (*vide* p. 220), but the blood in the living body is always alkaline. What the degree of alkalescence of normal blood is, has not been definitely determined; but it is probable that, like the temperature, it cannot be passed in either direction without disturbance of healthy nutrition. Experiments on animals with a view to reduce the alkalinity of the blood have resulted in changes in the tissues closely resembling the appearances found in the bodies of persons dying from scurvy. The primary change, then, in scurvy appears to be a chemical alteration in the quality of the blood, in the direction of diminished, or vitiated, or altered alkalinity.

Dr. Gaskell[1] has also shown that an alkaline solution excites contraction of the heart and of the smaller arteries, while acid solutions produce an opposite effect. It is, therefore, probable that variations in the degree of the alkalinity of the blood lead to errors in the circulation, and so effect a secondary influence on nutrition, as well as a direct one.

A consideration, however, of all that has been advanced on the subject leads to the conclusion that we are still ignorant of the precise changes in the fluids and tissues of

[1] *Journal of Physiology*, No. 1, 1880.

the body which precede and accompany the development of scurvy. And this notwithstanding there are 773 separate works on the disease and a still larger number of papers. Practically the following results may be stated:—In all scorbutic diets, such as salt meat, dried leguminous vegetables, rice, bread, &c., potash exists in small quantities. Scurvy presents a diminution of alkalinity of the blood. All anti-scorbutic diets contain a large amount of vegetables affording a large proportion of potash.

It is worthy of remark that there appears to be a pathological relation between scurvy, gout, and rheumatism. In scurvy there is diminished alkalinity of the blood owing to the withdrawal of alkaline bases supplied by vegetables, the result being an acute effect of acid on the tissues leading to textural degeneration. In gout there is diminished alkalinity induced by the positive addition of acid and acid salts in the blood leading to degenerative changes in tissues of low vitality. In rheumatism an abundant generation of acid seems to be excited by catarrhal influences.

Causes.—Scurvy was formerly supposed to depend chiefly on the use of salted provisions, but experience (such as that of the 75th regiment in Caffirland, of the English troops in the Crimea, at the siege of Paris by the Germans) leads to the belief that all insufficient, exclusive, or artificial diet, if long persevered in, will induce symptoms of the disease; and the sooner if the defects of food involve a loss of the just proportion of succulent vegetables with their salts of potash and azotised material. Dr. De Chamount[1] observes, 'Scurvy is likely to follow any material or prolonged disturbance of the equilibrium which ought to be maintained between the different articles composing a diet. But even when this equilibrium is kept up mere sameness of diet will after a time induce it.' Thus, it is the *absence* of certain constituents of the food, instead of the *presence* of noxious

[1] Dobell's *Year Book*, 1872.

material, from which scurvy arises. The effects of elevated temperature, malarious influences, extreme cold, night damp, impure atmosphere, especially from overcrowding, insufficient shelter, indolence, want of suitable exercise, fatigue, previous attacks of illness, especially malarious fever, syphilis, depressing passions, and continued absence of solar light, are powerful predisposing causes, and even in some instances there is reason to think may be exciting causes. Some authors have considered that dysentery exerts a peculiar predisposing influence, but although dysentery is often combined with scurvy there is no reason for presuming that dysentery predisposes to scurvy more than other debilitating maladies.

With reference to scurvy being caused by the absence of certain constituents of the food, instead of by the presence of noxious material, Parkes may be quoted, who said, men have been fed with an amount of nitrogenous and fatty food sufficient not only to keep them in health but to cause them to gain weight, and yet have got scurvy. This, as elsewhere noted,[1] I have seen exemplified on the return of native regiments to Bombay from service at Aden. Many of the men, and especially some of the native officers, were in especially good condition, and some of the latter were even too fat, and they were very much surprised when, from the unmistakable evidence afforded by the slightly tumid although not sore gums, they were requested to take a ration of lime-juice and sugar every day. The acknowledged good effects of lime-juice and fresh vegetables does not, however, warrant the conclusions that the absence of such agents is the only cause of scurvy, or that the presence of such agents will invariably cure scurvy. As before stated, experience appears to show that an insufficient or artificial diet, if long persevered in, will induce symptoms of the disease; unless indeed the insufficiency is so great as to produce starvation and famine

[1] The author 'On some Points Connected with Scurvy,' *Bom. Med. and Phy. Soc. Trans.*, 1882.

fever first. The number of persons suffering from scorbutic symptoms during the famine in Rajpootana (in 1869–70) was enormous, and this amongst the classes who were not actually victims of the advanced stages of want. Yet these people were perhaps consuming more fresh vegetable material at that time than at any previous period of their lives, their diet being supplemented by what I have elsewhere[1] described as the 'famine foods of Marwar.' Yet, notwithstanding the actual *addition* of fresh vegetable food, scurvy occurred with the scarcity.

The native troops stationed at Aden and Perim are very liable to scurvy, and this although the diet supplied to them, which has been supervised with great care, contains a sufficient quantity of anti-scorbutic material. The fact of scurvy occurring under such circumstances has been attributed to various causes (especially to the men selling their rations), but no satisfactory explanation has hitherto been adduced. Europeans who are exposed to similar climatic influences are rarely scorbutic. But an instance of this kind recently came under my observation at Aden, in the person of a European lady, who always took the usual amount of fresh vegetable material, and had not suffered from other ailment.

A remarkable development of scurvy, clearly unconnected with the usual cause, a deficiency of fresh vegetables, is afforded by the history of the Austrian garrison at Rastatt, in 1851, where 610 cases of scurvy occurred, principally during May and June, at a season of the year when fresh vegetables were abundant. Ziemssen gives eight recent examples, more or less similar to the above. The scurvy which attacked Burk's exploring party in Australia is also to the point. Here, according to Dr. Beckler, the medical officer of the expedition, there was no deprivation of fresh vegetables. There can be no doubt, however, that scorbutic symptoms will arise from insufficient diet in a shorter space of time if the defects of

[1] *Ind. Med. Gaz.*, 1870.

food involve an insufficiency of fresh provisions. It is probable that the immediate cause of scurvy is in most instances a deficiency of the potash salts of the vegetable acids in the food. But I do not endorse the assertion, which has been so confidently made, 'That an invariable antecedent of scurvy is a deficiency or absolute want of fresh vegetable food.' It is more than possible that scurvy does not always arise from the same derangement of general nutrition. If it does arise from the same derangement of nutrition, it is probable that such derangement may result from different causes. The usual predisposing causes of scurvy (previously mentioned, p. 466) when specially potent, or in combination, may produce the disease, either without any or perhaps with an inappreciable dietetic error, involving an infinitesimal loss of anti-scorbutic properties. It must be recollected that the individual predisposition to all diseases varies greatly, and that the individual predisposition to scurvy is strongly increased by malarious influences, dysenteric maladies, and syphilis: all affections common in the tropics. Even Ralfe, who regards scurvy as due to want of vegetables, admits that the absolute deprivation of vegetable food is not required to produce a tendency to scurvy, some persons being naturally disposed towards the disease and more readily and speedily affected by a temporary withdrawal or a diminished supply than others; these persons having what the older authors termed the 'scorbutic diathesis.' All the predisposing causes mentioned above do, however, lead to want of appetite and disorder of digestion, resulting in imperfect nourishment of the tissues and lessened power of withstanding noxious influences. In this manner only can those undoubted instances of scurvy be accounted for which have presented when there has been no lack, or at least no appreciable lack, of anti-scorbutic diet.

How scurvy originates in some instances is described by Leach, who remarks, 'A sailor goes from England to Calcutta,

and shortly after arrival is attacked with dysentery or intermittent fever, fractures a limb, or becomes syphilitic. He remains in India a short time, ships in an enfeebled condition, and lies up before the ship has been many days at sea. The berth he occupies, with very little change of clothing, is probably wet, his food scanty and unvaried, and his lime-juice or other anti-scorbutics served out irregularly.' Under such circumstances scurvy soon begins to colour the original disease, so that as a consequence the recovery of the patient is deferred solely on account of this scorbutic condition for weeks or months.

Classes Subject to Scurvy.—The male sex has been supposed to be a predisposing cause of scurvy, but this is not correct, as the fact of scurvy presenting more commonly in men than in women is explainable from the former being more frequently placed in those circumstances under which scurvy arises. A similar explanation of scurvy being most usually met with in men in the prime of life may be accepted. Under ordinary conditions scurvy is rare among women and children. Still, women in India of the poorer classes, who are indifferently nourished, are often found either latently or slightly openly scorbutic, and there is reason to believe that the stomatitis and aphthæ from which so many children suffer is due to the same cause. Infants have been known to become scorbutic when fed with milk from a scorbutic mother, and this occurs more or less patently or latently. Experience in Russia has also shown that children are by no means exempt from scurvy.

Scurvy has also been attributed wholly or partly to depression or low spirits in those isolated from society. Prisoners in Indian jails, although their dietary is varied and apparently sufficient, are frequently scorbutic, and the depression consequent on imprisonment has been regarded as the cause. The regularity of prison work, sleep, meals, the absence of hope, fear, excitement, amusement, the anxiety

regarding friends and family outside, no doubt cause mental depression in the majority of prisoners. Mental depression lessens the activity of all functional processes, thus rendering the system more likely to be affected by deleterious influences. Thus a dietary which is sufficiently anti-scorbutic for a free man is not sufficiently so for a prisoner—the proof being that when scurvy occurs among prisoners the immediate step has been an increase of anti-scorbutics in the dietary.

Explorers and pioneers in all countries have been especially liable to scurvy. It was very prevalent among the gold-diggers in California, and is still so among pilgrims in the East, who, like gold-seekers, incur great fatigue on very indifferent diet.

Prevalence of Scurvy in India.—Since the prophylactic properties of vegetables have been appreciated scurvy among Europeans in India, except in times of war, has ceased to assume the formidable proportions of former days. Nevertheless, Dr. Hewlett, Sanitary Commissioner, Bombay, in a report (1884) 'On Enteric Fever in the Bombay Army,' remarks on the existence of the scorbutic taint throughout the European troops, often not so bad as to attract attention, and regarded by some medical officers as malarial cachexia. The Indian military surgeon attending native troops is at times called upon to treat scurvy in his regimental hospital, especially if the men have been on active service, or in the Bombay Marine Battalion, or at Aden, or elsewhere out of India. The Indian civil surgeon is called upon to treat scurvy most frequently, if his duties are connected with the presidency or coast hospitals, or with the merchant shipping in harbour. But the cases are comparatively few, and of a less serious nature than those presenting even thirty years back and within the author's recollection. But notwithstanding this, the scorbutic diathesis is a *very prevalent condition* among the natives of India, more especially in some parts of Western India where scurvy may be rightly regarded as

endemic; sometimes, in consequence of scarcity or famine, assuming an epidemic intensity.

Wherever, as so frequently occurs in Western India and even the North-West Provinces, there is a soil highly impregnated with saline material, especially on the borders of the semi-desert districts, and on arid and sandy sea-coasts, there the scorbutic diathesis will be found to prevail. It may not, and indeed generally does not, display itself by the swollen spongy gums, the petechiæ, and the indurations of the confirmed disease. On the contrary, it usually remains more or less hidden or *latent*, the manifestations being perhaps the very slightest redness of some part of the gums, which however must be sought for, as it would not be noticed by the patient. Or perhaps there may be trivial-looking sores on the mucous membrane of the mouth or tongue, similar to those arising from simple dyspepsia. Or the face may show a little puffiness under the eyes as the only sign, or the patient may complain of vague wandering rheumatic pains. Otherwise the manifestation of latent scurvy may be that some sore or ulcer will not heal.

When treating cases of so-called recurring malarious fevers, when treating spleen diseases, secondary syphilis, chronic diarrhœa and some other affections, I used to find in Rajpootana, and especially in Marwar, that it was necessary first to attack the underlying scorbutic condition; and remedies calculated to effect this were often insisted upon when there was really no outward evidence of scurvy which can be described. I believe, however, that the latent scorbutic condition may be present without manifesting itself by any signs or symptoms, and that when so present it influences all diseases which may affect the individual, obscuring and masking and preventing the cure of those ailments which belong to medicine, and aggravating all surgical complaints. The probability, therefore, of the existence of this scorbutic taint should never be lost sight of,

forming, as it does, one of the most dangerous constitutional defects, whether occurring in the civil population or in the military forces. Thus it has happened that while a Sanitary Commissioner is drying the ink with which he congratulates himself on a small death ratio, scarcity occurs, the latent scorbutic diathesis exerts its sway, and the people die; not from scurvy, but from other maladies aggravated by the latent scorbutic constitutional taint. Again, a Government or a Commander-in-Chief seeing a corps presenting a low death-rate issues orders for that corps to proceed on service or to some other locality—Aden for instance. After a short period, as a consequence of fatigue or of change of diet and climate, the scorbutic diathesis becomes developed, and, very much to the surprise of the authorities, the low death-rate is succeeded by a heavy sick list.

The existence of latent or incompletely developed scurvy has also been noticed in England, especially by Drs. Eade[1] and Buzzard,[2] the latter pointing out that in consequence the true nature of an ailment is often overlooked. Ralfe[3] also states, 'In many instances of persons supposed to be gouty I have suspected them to be really suffering from an incompletely developed form of scurvy, and have noticed an almost immediate alleviation of the symptoms by recourse to lemon-juice.' Ralfe also mentions, he makes it a rule to examine the gums of all patients at the London Hospital, adding that the scorbutic condition is not confined to the lower classes.

The causes of the underlying scorbutic condition which is believed to be so prevalent in Western India are not far to seek. It has been mentioned that latent or hidden scurvy prevails chiefly where there is a soil highly impregnated with saline matter, and this whether such saline

[1] *British Med. Journ.*, Nov. 19, 1881.
[2] Reynolds, *System of Medicine*, p. 745.
[3] *Morbid Conditions of the Urine*, p. 73.

material is common nitrate of potash, or that combination of salts known as 'reh.' It is in such saline, damp, low-lying localities that the production of vegetables is at a minimum, as very few things will grow in such a soil. But in estimating the reasons why scurvy is so prevalent in Western India various other predisposing causes besides the absence of fresh anti-scorbutic vegetables must be taken into consideration. These are, especially in the more northerly districts, elevated temperature by day, and cold nights leading to overcrowding during the latter period of the twenty-four hours, damp, malaria, fatigue, bad water, and the absence of meat from the dietary, poverty, all of which are in full operation amongst natives. To a certain extent the same causes operate on Europeans, whose appetite is generally at the minimum in the hot weather, causing them to eat less meat at exactly the season when fresh vegetables are least plentiful. Moreover, Europeans, especially up-country where the hot winds prevail, often subject themselves to another predisposing cause. The hot winds, and the light also, are shut out of their bungalows for the sake of coolness. That absence or deficiency of solar light is a powerful predisposing cause of scurvy in the Arctic regions is rendered evident by the writings of Kane, McClintock, and other Polar travellers, and there is every reason to presume that absence or deficiency of solar light exercises a similar influence in one zone as it does in another. It does so on the vegetable world; a potato sprouting in the dark, whether in India or in England, putting forth unduly lengthy and blanched shoots, which moreover turn in the direction of any ray of light as if requiring that stimulus. Natives also are to a certain extent doubtless affected by absence of solar light, many of them living in huts with only one small door, which is often closed for hours in the middle of the day to keep out hot winds or dust.

Symptoms.—A minor degree of the degeneration con-

stituting scurvy may exist (like the poisons of syphilis and hydrophobia) for an indefinite period without any appreciable symptoms. 2. It may cause what at first appears to be simple anæmia. 3. It may manifest its presence by simply delaying convalescence from other diseases, by causing a slight bruise to become an ulcer, or by retarding the healing of sores, such sores often not presenting the usual spongy appearance and propensity to bleed of the confirmed scorbutic ulcer. 4. Such maladies as the Delhi sore, the Scind boil, the Gwalior ulcer, the Aden boil, the Surat boil, the Burmah boil, the Baghdad boil, may be frequently traced to those conditions under which scurvy arises. 5. Scurvy sometimes developes itself by such premonitory symptoms as malaise, wandering rheumatic pains, a little puffiness under the eyes, ulcers of the mouth, and soreness of the tongue, the gums being unaffected and no other symptoms of scurvy being present. The two latter conditions, more or less preceded or accompanied by a sub-acute inflammatory condition of the mouth or tongue, occurred in my knowledge to certain troops in the Persian Gulf during the Persian war of 1857, to a detachment of Madras sappers and miners stationed at Butcher's Island in 1878, and to certain troops of the Malta expeditionary force of 1878 when stationed at Cyprus. Stomatitis and aphthæ occurring to children as a development of scurvy has already been mentioned (p. 469). 6. Natives suffer considerably from a chronic dental suppurative periostitis, which is mostly scorbutic. 7. Debility and palpitations of the heart with dropsical swellings, especially of the abdomen, may exist in badly fed people, apparently partly as the result of starvation and partly as the result of scurvy. This was exemplified during the famine in Rajpootana (1868–69), and after the famine in Western India, especially in Goozerat in 1878. 8. A diarrhœa with chocolate-coloured stools has been noted as the principal accompaniment of a minor scorbutic taint, and may be the

only manifestation of the scorbutic condition. 9. Scurvy may be manifested by a chronic conjunctivitis. 10. It may show itself as, or at least develope gout. 11. It may appear as 'beri-beri' (*vide* p. 55).

When scurvy passes that latent period when no recognisable symptoms are presented, there is usually an initial period of indefinite duration—weeks or months—during which the patient may suffer more or less from malaise, depression, unaccountable debility, drowsiness, apathy and indolence of mind, great sensibility to low temperature and wandering rheumatic pains, accompanied or followed by soreness of the gums, sometimes so slight as to be unnoticed by the patient; sometimes presenting, at others not presenting the reddish line described below. Or there may be a tumid condition of the gums without sensible soreness. The gums, however, may become affected without the patient noticing any of the symptoms noted above as belonging to the initial period. In a minor number of cases there is at first no affection of the gums, infiltration of other tissues as described below taking the lead. The reason why the gums are so frequently primarily and so severely affected, does not appear to lay in any peculiarity of structure, but seems due to the fact of the gums and teeth being constantly in use, and to the gums not being covered with a thick skin as the hands and feet, and hence being liable to irritation from contact with material which would not affect them if the system were in a healthy condition. It has been intimated that there are cases of scurvy in which the gums are not primarily attacked. Similarly, where teeth are wanting the gums often remain sound. The same occurs in childhood and old age when there are not any teeth. But usually, if the disease is unchecked, the gums becoming affected grow worse, presenting a dark reddish or purplish margin at the teeth from a line to a quarter of an inch in breadth; the gums between the teeth forming little knobs or projections.

With reference to the condition of the gums, I now have to make an observation which I regard to be of importance with reference to the earlier symptoms of scurvy. Many natives show blue marks or patches on the gums, yet being apparently in the best of health. Is this a manifestation of incipient scurvy? I believe not. It is, I think, simply a deposit of pigment. But when the troops were examined previous to leaving for the Malta expedition and to Afghanistan, some medical officers were inclined to reject men showing these blue discolorations. This however, after consultation with the then Surgeon-General, Sir Guyer Hunter, was overruled. And we were induced to take this course from never having found any of these blue patches on the gums of Europeans. The coloured patches I refer to are quite a different thing to the blue line at the edges of the gums where they come in contact with the teeth, and which blue line is described as a symptom of scurvy by Aitkin, by Chevers, and by most other authors on the subject. The matter is one of importance, for in consequence of these pigmentary patches being regarded as a manifestation of scurvy, men may be rejected when they should not be. It is moreover, so far as I am aware, a matter which has not been mentioned in any book or treatise on the subject of scurvy.

If the disease progresses, the gums become more swollen, spongy, sometimes livid and bleeding at the slightest touch, while the teeth grow loose, and there is an offensive sour fœtid odour. Both the appearance of the gums and the odour are very different from that caused by mercury. It is astonishing the number of natives who go about their avocations with the gums more or less scorbutically affected as above, but otherwise in apparent good health. As soon as or before the gums become greatly affected other symptoms present. There is dull pain in the limbs, often cardiac palpitation, and nearly always shortness of breath on exertion. Some authors state 'the chest is generally free,' but in the

cold season, bronchitis and pneumonia are not uncommon, and there is usually some degree of cough and engorgement of the lower lobes of the lungs. The countenance becomes of a dull leaden or sallow hue, and appears bloated, while the cheeks especially present a puffy aspect from the swollen gums within. The odour from the mouth becomes more offensive and fœtid, and may be aggravated by aphthous spots, by salivation, by sloughing, or even by necrosis of some part of the jaw. The teeth are of course loose, and may fall out. The tongue is generally pale, often clean, and always tremulous. Petechiæ may now occur on various parts of the body, and these are similar to *purpura*. They consist of isolated spots, varying in colour on the white skin from a bright to a livid red, and in size from that of a pin's head to that of a shilling. These petechial spots are not so vividly apparent on the dark skin, and the colour is much darker. They are very slightly elevated, and do not disappear on pressure. Sometimes small nodules appear, which have been termed *Lichen scorbuticus*, or *acne scorbuticus*; at other times small vesicles present, known as *Herpes scorbuticus*. Either at the same time or after the appearance of petechial spots, the characteristic extravasations or effusions of scurvy present. These effusions are of a hæmorrhagic and fibrinous nature, and principally occur in the lower extremities. They present externally as extensive blotches or ecchymoses of irregular form, occasionally striped or streaked in appearance, and feeling more or less hard to the touch. Any part previously injured is very likely to be thus affected. The muscles of the thighs and legs are often attacked. Effusion may also take place into the loose cellular tissue about the tendo Achillis and hollow of the knees. The joints may also be swollen by effusion into their cavities. The joints being thus rendered swollen and stiff and the muscles rigid and painful, the patient is unable to move the limbs, which usually remain in a more or less semi-flexed condition. The

swelling about the legs must not be regarded as dropsy, as it does not pit on pressure in the characteristic manner of dropsical effusions. There may also be painful node-like swellings from effusion between the periosteum and bones, especially of the tibiæ, in persons who have suffered from venereal. The orbit, scalp, and skin of the face, are often affected. Sometimes over any of these effusions large vesicles form and fill with sanguinolent fluid (*pemphigus scorbuticus*), and these may become ulcers as presently described. Effusions sometimes take place in the fauces, pharynx or larynx, the laryngeal implication being dangerous as it may lead to œdema of the glottis.

When scurvy is thus fully developed, the skin generally is very dry, and may be thrown off by a desquamation of a branny character, or in larger scales. Not only are all the processes of repair arrested so that wounds of any kind will not heal, but the skin is so sensitive that slight pressure, as of one limb on the other, particularly on the inner surfaces, may produce a bruise. Scratches become foul ulcers, old cicatrices open afresh, and previously fractured bones may become disunited. In severe cases, the ribs may be detached from their cartilage.

As scurvy progresses, there is usually great emaciation, although this may be to a certain extent masked by the swellings characteristic of the condition. Passive hæmorrhages are also liable to take place from the nose, bowels, ears, kidneys, bladder, or lungs. There is anxiety, sleeplessness at night, the pulse becomes small and frequent, and the temperature of the skin is lessened, although on this point thermometric readings are still required. The bowels are sometimes constipated throughout, but more frequently there is looseness, and often diarrhœa or dysentery, the evacuations being intolerably fœtid. A diarrhœa, with chocolate-coloured stools, has already been noted (p. 474) as a manifestation of scurvy. Very frequently also diarrhœa assumes

a hæmorrhagic character. The urine is small in quantity, containing abundant chlorides; urea, phosphates, and potash, especially during the later stages, being deficient. It is usually free from albumen. During convalescence there is an increase in the quantity of the fluid and of solid matter. As regards the appetite, it is generally bad or capricious, with no great thirst, but with a craving for acids; but sometimes good and even voracious, until a very advanced stage of the malady. Some scurvy patients desire just that kind of food which they should not have. But this craving after improper diet does not appear to be so frequently developed in tropical as in Arctic scurvy, for Hale[1] tells us, 'Those who had it seemed determined to die, for against all reasoning and advice, they would have salt pork in preference to fresh game,' and 'They would go any length to obtain improper food, notwithstanding they were well aware death must follow this contumacious course.'

Complications.—In all severe cases of scurvy the patient is subject to sudden and sometimes fatal syncope on the slightest exertion. *Embole*, derived from *thrombi* which form in the right auricle or its appendage as a consequence of the feeble circulation, is also a source of danger. As before mentioned, epistaxis or hæmatemesis, or hæmaturia, or œdema of the glottis, may all occur and prove fatal. Hæmorrhage from the respiratory organs is more rare. More or less acute congestion of the lungs, or pneumonia of a croupous character, may occur, especially in the cold season. Pericarditic or pleuritic effusions sometimes take place, the pericardium being most frequently affected. A previous rheumatic condition is considered favourable to such developments. Intermeningeal hæmorrhage, giving rise to pain in the head, dulness, stupor, or convulsions, is an occasional complication or sequel. But the most common termination is by exhausting diarrhœa or dysentery. Unless some effusion or

[1] *Life with the Esquimaux*, p. 22.

hæmorrhage occurs implicating the brain, the intellect remains clear to the last.

The *scorbutic ulcers* which accompany or follow scurvy often result from the vesicles which have been mentioned as arising from the petechia or extravasation. They vary in size from that of a shilling to the palm of the hand. They are usually surrounded by a broad discoloured zone. The surface is foul, with shreds of disintegrating tissue streaked with blood, or there is a spongy bleeding surface with great tendency to sloughing. Sometimes these ulcers extend deeply, open blood-vessels, and occasion alarming hæmorrhage. Or they may occasion such injuries to muscles and tendons that permanent contractions or even dislocations are the result. In some cases of obscure scurvy ulcers may be the principal or only manifestations of the disease, but they are usually attended by some other scorbutic symptom. Persons of scrofulous constitution if becoming scorbutic are exceptionally liable to the ulcerative form of the malady, and in scrofulous children the nails may become affected (onychia scorbutus). *Cancrum oris* has also in some instances appeared, consequent on a scorbutic taint. Scorbutic ulcers have prevailed to a great extent among troops, and in prisons, although not always recognised as such.

Hemeralopia, or night-blindness, having been found associated with scurvy, has been thought due to the scorbutic condition. Scorbutic forms of conjunctivitis have already been referred to, and a scorbutic iritis has been elsewhere described. The fact is, there is scarcely any malady which may not arise in the scorbutic system, and although scurvy may not be the exact cause it is the underlying and predisposing condition. A scorbutic taint, either latent or declared, appears to exert a special and peculiar influence in exciting any disease to which there is the slightest tendency, hereditary or acquired; and especially gout, to which (*vide* p. 465) scurvy seems pathologically

allied. The triune complication of scurvy, malarious or heat cachexia, and syphilis, is probably the most intractable diseased condition known. In short, as the scorbutic taint renders the system an easy prey to almost any other disease, and as it aggravates all subsequently contracted maladies, the scorbutic condition itself is rendered more intractable by such maladies.

Post-mortem appearances.— Body emaciated, but not always looking so, owing to effusions in the tissues. Gums œdematous, spongy, and occasionally ulcerated. The thighs and legs feel more or less hard, and the cut muscles have a streaky appearance, from the effusion of fibrinous material between the striæ. Some of these effusions are clearly defined, others not so; some are soft, others harder; some darker in colour than others, the oldest effusions being most defined, hardest and darkest. In severe cases the ribs may be found loosened, or even detached from the cartilages. Occasionally there are ecchymotic spots on the heart, pleuræ, or pericardium, and often more or less congestion or œdema, and sometimes hepatisation of the lower part of the lungs. The liver is often healthy, but sometimes presenting fatty degeneration. Spleen usually friable, pulpy, but sometimes sound. Effusions in the pancreas have been occasionally observed. Kidneys generally healthy, unless albumen has been passed, when they may be found degenerated. Ecchymosis of the external coats of the intestines, especially of the lower part of the small bowels. If there has been diarrhœa, the mucous coats may be red and congested. If there has been dysentery, there may be ulcerations, which however have generally lost their characteristic dysenteric appearance, being more ragged, more ill-defined, and not so much excavated as the purely dysenteric ulcer.

Under the microscope the blood-corpuscles appear shrivelled, and fatty changes may be detected in the se-

creting cells of the liver and kidneys, and in the muscles generally.

Diagnosis.—From purpura. The terms *purpura simplex*, *purpura hæmorrhagica*, &c., have been applied to different stages or varieties of scurvy, and there is reason to believe purpura and scurvy are not distinct affections. Purpura is a diseased condition in which circumscribed effusions of blood take place into the upper layers of the cutis, occurring with or without certain constitutional symptoms, appearing in the course of various diseases of which scurvy is one, and attended at times by hæmorrhages. Purpura has been thought due to an alteration in the nutrition of the coats of the minute blood-vessels, which renders them unequal to the strain of arterial pressure; or *secondly* to an alteration of the blood itself in the direction of excess of water and salts. It is, however, most probable that the alteration in the coats of blood-vessels (if any) depends on changes in the blood, so that both scurvy and purpura are probably due to somewhat similar causes. In purpura, owing undoubtedly to rupture of capillaries, blood finds its way into the meshes of the connective tissue. Then first the serum is absorbed, and afterwards the hæmatin, which passes through various colours, blue, green, yellow, as if effused from some other cause.

Purpura appears as isolated spots, chiefly on the lower extremities, varying at first from a bright red to a purple red, and which do not fully disappear on pressure. The spots are irregular in shape, and differ in size from that of a pea or bean to a pin's head. The smaller spots are termed *petechiæ*, the larger spots are called by some *ecchymoses*. They do not itch, they are not scaly, there is no tendency to discharge, and they never desquamate. Their appearance is sometimes accompanied by symptomatic fever, at other times there is no appreciable fever.

Purpura is said to differ from scurvy as follows. The

spots of purpura, although very similar in appearance to the ecchymoses observed in scurvy, rarely exceed the size of a shilling, while those of scurvy are often much larger. Purpura frequently occurs in persons who have not suffered from any deprivation of fresh vegetable food, or from privation, or from sameness in diet, while scurvy ordinarily does not do so. *Thirdly*, purpura is not accompanied by affection of the gums, painful swellings, or large ecchymoses of the skin. *Fourthly*, there is an absence in purpura of the severe cachexia of scurvy. *Fifthly*, purpura is presumed to be due to an alteration in the coats of the small blood-vessels, while scurvy is due to an alteration in the blood itself. Lastly, purpura, unlike scurvy, does not improve under anti-scorbutic remedies.

It may, however, be observed that the small spots presenting in scurvy cannot be distinguished from purpura; that scurvy occurs in persons who have not suffered from any appreciable error of diet (p. 468); that scurvy shows itself without affections of the gums or painful swellings (p. 475); that scurvy may be present without cachexia (p. 466); that it is by no means certain purpura is due to alterations in the blood-vessels, and that it may be due to alteration in the blood itself (p. 482); that purpura is not always benefited by anti-scorbutic remedies, is generally relieved by measures which tend to strengthen the system by improving nutrition. If, therefore, it may not be confidently stated that scurvy and purpura are the same disease, it may at least be asserted that they are allied in character, and that purpura occurs in the scorbutic condition, as a consequence of the same alterations of the blood which produce scurvy, for, as before stated, it is impossible to distinguish between the small petechiæ of scurvy and those of purpura. Even those denying the sameness of diseases admit the similarity of the spots.

In my humble opinion, the petechiæ of the earlier stages of scurvy, the petechiæ of purpura, the so-called specific erup-

tions of enteric and of typhus, are simply modifications of purpura, and are all due to the same cause, viz., alterations in the blood consequent on a diseased state of system. It is admitted that purpuric spots occur in the course of various diseases, and it is also admitted there is no abrupt line between any varieties of purpura (Sparks and Sangster). The different varieties of purpura called *petechiæ*, *simplex*, *ecchymoses*, *vibices*, *hæmorrhagic*, differ more from each other than do the so-called specific eruptions, or the scurvy ecchymoses from purpura. The ordinary purpuric eruption has been described as small isolated spots, with colour varying from bright red to dark purplish red, &c. (*vide* p. 482). In scurvy it is stated by Leach there are usually a number of spots and patches very much like those of purpura scattered indifferently about the lower limbs. In my experience such ecchymoses are usually the first stage of scurvy, and they cannot be diagnosed from purpura. The enteric eruption is described as rose-coloured spots, about the size of a pin's head (as some purpura spots are), slightly raised and pointed (as some purpura spots are), well-defined (as some purpura spots are), and disappearing on pressure (which they do not always do). In fact, as Dr. Broadbent states, there is nothing very peculiar in the spots of typhoid to distinguish them from others. The typhus eruption is described as small dusky brown red spots (as some purpuric spots are), not raised (as some purpuric spots), sometimes indistinct (as some purpuric spots), not disappearing on pressure (as purpuric spots). And moreover, it is stated petechiæ may be present. In short, these eruptions of typhoid and typhus, purpura and scurvy appear identical.

The diagnosis of scurvy from *anæmia*, when neither affection of the gums, nor petechiæ, nor swellings are evident, is more important. The pallor of scurvy is more livid than the paleness of anæmia, while in the latter there is at first less physical depression, and generally no rheumatic muscular

pains. But as Dr. Hewlett[1] observes, the scurvy taint so manifest among Europeans in India is often mistaken for malarious cachexia.

Prognosis.—Ordinary scurvy may be said to be limited by the duration of the causes which produce the disease, and if no complication exists, recovery from scurvy is usually rapid and thorough. Sometimes, however, when sufficient care is not taken to ensure the proper diet, or when a syphilitic or a malarious taint exists, recovery is slow, the gums remaining affected for months, although no other indication of scurvy may be present. This I have observed amongst native troops, who probably did not use all the anti-scorbutics ordered for them, although perhaps taking sufficient to prevent the scurvy growing worse. But, as mentioned above, although recovery from scurvy is usually thorough, it may leave the gums indurated for life, while the parts into which effusion has taken place may remain indurated, and the joints remain stiffened. In the advanced stages of scurvy the probability or otherwise of recovery is in exact proportion to the importance of the organs implicated. Dysentery is the most usually fatal termination. But this occurs in the East in consequence of the liability of the inhabitants to abdominal fluxes, which liability is increased by scurvy. Thus although more persons succumb to dysentery complicated with scurvy than to any other complication, the occurrence of thoracic disorders is perhaps even more dangerous. The passage of albumen during scurvy if persistent is always a grave omen.

Treatment.—Murray, Harty, and others of the older writers on scurvy, thought some cases at the onset were benefited by active measures, as bleeding and saline purgatives; but modern experience condemns this treatment, and defines the correct path as almost purely dietetic, on the principle that want of fresh provisions is the chief

[1] *Report on Enteric Fever in the Bombay Presidency*, 1884.

ostensible or hidden cause. The use of fresh vegetables and of fresh meat is at once the great preventive and curative means, and where these are deficient limejuice, as recommended two hundred years back by Woodhall, and again at a later period by Lind. Of lime or lemon juice, from five to six ounces should be taken daily in any stage of the disease, mixed with pure water and sweetened with sugar according to taste. Freshly made animal broths should also be given, if possible alternating with Liebig's fresh meat soup. This may be supplemented by several eggs daily and milk *ad libitum*. Solid animal food should also be given if it can be masticated, and if there is no dysentery. As adjuvants, fruits, such as oranges, pummaloes, scraped pears and apples, mashed potatoes, molasses, cocoa, pickles, vinegar, onions, nopal, and especially all the cruciferæ (as broccoli, kale, cabbage, turnips, mustard, cress, watercress, radishes, spoonwort or scurvy grass) will be most beneficial. As before mentioned (p. 463), Dr. Garrod, Aldridge, and others have shown it to be very probable that the vegetables, named as possessing a reputation as remedies for scurvy owe their good effects to the salts of potash they contain. Hence the value of cresses and uncooked vegetables, as raw potatoes, much of the salts being dissolved out in the process of boiling. Dried and compressed vegetables are not spoken of favourably, and it is probable that some change or decomposition, particularly of the citrates they contain, takes place in the process of drying and preserving. When debility is marked the recumbent posture must be maintained, and any sudden motion must be especially guarded against, or death by syncope may occur. Claret may generally be allowed, and if there is great prostration of strength, spirits (of which whisky is the best) and malt liquor may be allowed. The natives generally take the spirit known as *arrack*, which if pure is as good for the purpose as whisky.

As regards medicine, Kane in the Arctic regions found salts of iron beneficial; but I believe quinine or arsenic are the best remedies, the latter if there is no bowel complaint, the former if there is. If aperients are required, which is comparatively seldom the case, infusion of tamarinds, cream of tartar, or sulphate of soda may be used, but great caution should be exercised in giving aperients on account of the existing tendency to bowel affections. Acute pains may be relieved by morphia or chloral cautiously given; and difficulty of breathing by diffusible stimulants as ether, ammonia, camphor. Ulceration of the gums requires astringent gargles of alum, decoction of bark, port wine, strong tea, or still better chlorate of potash. When purging occurs tincture of catechu may be given with advantage. If diarrhœa persists a milk diet is advisable, and syrup of bael should, if procurable, be taken.

The value of meat as an anti-scorbutic has been already alluded to. But it is necessary the meat should be quite freshly killed, as certain chemical changes occur after a few hours, rendering it less anti-scorbutic. Kane states the fresh meat of the walrus proved both preventive and curative. If meat cannot be masticated, Liebig's raw meat tea is advisable. If fresh meat cannot be procured, Liebig's extract of meat is to be recommended, as it contains in abundance and in a condensed form the salts of potassa, which are required.

The value of milk as an anti-scorbutic has been alluded to, and it may be taken *ad libitum* by all scorbutic patients. The anti-scorbutic properties of milk seem to be proved by the fact that thousands of children are fed on condensed milk alone without getting scurvy. Scorbutic diarrhœa will often yield to a milk diet, aided by a reasonable amount of fresh vegetable food. Ships should be supplied with condensed milk in addition to limejuice.

The *prevention* of scurvy entails attention to general and personal

hygiene with reference to all the generally predisposing, but as there is reason to infer sometimes exciting, causes enumerated at p. 466. The prophylaxis of scurvy, so far as dietary is concerned, consists in the due admixture of vegetable and animal food in the daily meals, and in cases where the former cannot be obtained, and when the latter cannot be procured, in the use of limejuice, tamarinds, compressed dried apricots from Affghanistan (alu Buchara), citric acid; or potash if nothing else can be obtained, which Henderson procured from gunpowder, and Garrod suggested might be got from tobacco.

It is extremely difficult to obtain anti-scorbutic remedies in India. I am not acquainted with any indigenous vegetable of the class the vernacular designates 'turkarry' (meaning garden vegetables) which has anti-scorbutic properties. And almost universally, speaking now of the Mofussil, neither potatoes nor any other English anti-scorbutic vegetables are cultivated. This want of anti-scorbutic vegetables led me to usually recommend the young tops of the gram plant (*Cicer arietinum*) which are sub-acid and have been taken with advantage. I have also seen it stated in books that the custard apple (*Annona reticulata*) is anti-scorbutic. This, however, I scarcely believe, and if it were so, it is practically useless, as it can only be obtained in its short season. Limejuice or tamarind infusion may of course be substituted for vegetables, but I have little confidence in limejuice after it has been kept any length of time in a tropical climate. It certainly deteriorates both in appearance and flavour, and I believe that the spirit added to it under the idea of preserving it (as is done by the Commissariat Department) has a totally opposite effect. Limejuice to be efficacious in scurvy must in a hot climate be fresh, and so must tamarinds. It was this difficulty in obtaining anti-scorbutics which led some few years back to the use of Kokum (*Garcinnia purpurea*) for the troops at Aden, where a certain proportion, half an ounce, is supplied by Goverment to every sepoy daily. This, however, does not suffice to keep them free from scurvy, neither does the issue of Commissariat limejuice and sugar suffice to do so, or to check scurvy when it has once appeared. I was, therefore, led to recommend what I believe will prove to be the best anti-scorbutic in India. This is the dried unripe mango cut from the stone and dried in the sun. It is known in Bengal chiefly as 'Amchur,' in the North-West Provinces as 'Am-ka-chitta' and in Western India as 'Ambosee.' It used to be eaten in the jails of Rajpootana, and by analysis made by Dr. Murray Thomson, the Chemical Analyser at Roorkee, it has been found to contain proportionally double the quantity of citric acid and malic acid found in fresh limejuice.

I have also to mention that I have read in several old works—non-professional—that the fresh milk of the cocoanut is a preventive and curative agent. I have no experience of this myself, never having, when

in practice, been in a part of the country where fresh cocoanut milk could be conveniently obtained. According to Dr. Lyons, Chemical Analyser, Bombay, fresh toddy contains 5·6 per cent. of alcohol three hours after being drawn, rising to 10 per cent. ten hours afterwards.

In a paper entitled 'Some Points connected with Scurvy,' read before the Bombay Medical and Physical Society in 1882, and published in the Transaction for that year, I made the following propositions:—

1. The great prevalence of latent scurvy in Western India.

2. That scurvy may exist without manifest symptoms of its own, yet aggravating and retarding the cure of other maladies.

3. That scurvy may exist, the individual being apparently well.

4. That insufficient diet will cause scurvy even if fresh vegetable material forms part of that diet; but that scurvy will be developed more rapidly if no vegetable material is taken.

5. That deficiency of solar light is a powerful predisposing cause of scurvy.

6. That there is no Indian indigenous anti-scrobutic vegetable.

7. That limejuice deteriorates and loses its anti-scorbutic properties by keeping in a hot climate.

8. That dried unripe mango is the best and cheapest and most convenient form of anti-scorbutic.

9. That blue pigmentary deposits on the gums of natives are not evidence of scurvy.

SCORBUTIC ULCERS.—It has already been stated that trivial injuries in those affected with scurvy frequently pass into foul and ill-conditioned ulcers having a strong tendency to phagedenic sloughing. These, sometimes opening arteries, render amputation necessary, or produce so great injury of muscles and tendons that permanent contractions result.

These ulcers have prevailed to a great extent among Indian troops employed in Burmah and elsewhere, and Mr. Ward formerly described an acute and chronic form. They are always attended with great debility, impaired appetite, white dry and loaded tongue, insomnia, quick pulse, and other signs of constitutional irritation, while there will generally be spongy gums, loose teeth, maculæ, or other symptoms of scurvy present. But the scorbutic ulcer may be the only evident manifestation of the scorbutic diathesis. The treatment consists in the employment of anti-scorbutic remedies; and locally in the application of dilute nitric acid, charcoal poultices, tincture of myrrh lotion, or decoction of cloves or nutmeg leaves, as recommended years back by Geddes. If a phagedenic condition presents, the application of strong nitric acid may be advisable.

Scorbutic nodes, previously referred to, are also common, and for these the iodine paint or tincture, applied externally, is a good local remedy, or several coats of white-lead paint, and bandaging.

CHAPTER XXVIII.

SKIN DISEASES.

ALTHOUGH all forms of skin diseases are seen in India, the most common are: 1, the various forms of Tinea; 2, Pityriasis; 3, Scabies; 4, Lichen tropicus; 5, Leucoderma; 6, Morphœa; 7, Fibroma.

TINEA.—There exist in the East, in different places, vegetable parasitic eruptions, apparently differing but really the same, and which have been variously styled 'China,' 'Indian,' 'Burmese,' 'Malabar,' Tokelan, and other ringworms. As Dr. Colan observed, 'As all the vegetable productions of the tropics surpass in luxuriance those of cooler regions, so there may be a greater luxuriance of these parasitic growths in different localities which have led to a variety of names.' Fox and Farquhar[1] remark, the time has come for the use of some common term for the various locally-named ringworms, and they suggest *tinea circinata tropica.*

There are two special forms of tinea or ringworm; the ordinary type which occurs on the head, especially of children; and in India an even still more common type, which occurs on the body. *Tinea tonsurans* presents in its earliest form as a little redness or scurfiness of some spot on the scalp which usually escapes notice, or if seen is regarded as unimportant from some other cause. Then in two or three days there are circles of minute pimples which also may not

[1] *Endemic Skin Diseases of India.*

be recognised until, in the course of a few hours, they become minute vesicles. These break, and discharging their contents produce a thin scab, which may be mistaken for scurf. But fresh circles of pimples and vesicles form on the outside of the first crop, the disease spreading in circular-shaped patches; and it is only at this time that many children whose hair is thick are brought for treatment. As the malady goes on partly from the discharge consequent on the eruption, and partly from the discharge induced by scratching, larger and thicker scabs form. Neglected ringworm may thus involve nearly the whole of the scalp, the latter stages being very similar to 'scald head,' or *favus*. There is, however, a peculiar condition of the hairs in the part affected which serves to distinguish ringworm from any other head affection. With a magnifying-glass and plenty of light the hairs over the affected spot appear as if rubbed off close to the scalp, the short portions remaining looking dry, lustreless, brittle, bent or twisted, and often split, or running in a line different to that of the healthy hairs, and affording a fancied resemblance to a stubble field. A drop of chloroform turns these hairs an opaque yellow, while it has no effect on healthy hairs. The hairs thus broken off are in reality dead, and when attempts are made to extract them they often break again, the point of rupture looking ragged. When the root comes away and is placed under the microscope, the distinctive fungus may be recognised in the shape of bright round clearly defined cellular bodies, about $\frac{1}{7000}$ to $\frac{1}{5000}$ of an inch in diameter, collected in chains or groups, and known as the *trichophyton tonsurans*. The most minute redness or scurfiness of the head of a child should always be regarded with suspicion as the possible commencement of ringworm. In such cases examination with a strong glass will often show either minute vesicles, or at a later stage lighter-looking portions of hair-shafts which have escaped observation by the naked eye.

Treatment.—In a case of *suspected* ringworm the head should be thoroughly shaved for an inch all round the part, the head should be washed with carbolic soap twice daily, and the child should wear a skull-cap. Then if ringworm is diagnosed, the great object is the removal of the diseased hairs, which should be extracted carefully one by one by a pair of broad-nibbed forceps. Unless this is done very gently, but at the same time firmly, the hairs will break, and the roots remain. The hairs removed should be burnt at once. Then every particle of scaliness should be washed away with carbolic soap and water. Then strong vinegar or alum water (four drachms to the ounce) or ink may be applied to the part affected. Ink is a popular and useful remedy, the good effects resulting from the iron and tannin it contains. Whatever remedy is used should be gently rubbed on the scalp with the finger so as to insinuate it into the holes from which the hairs have been plucked, and in which the fungus vegetates. The application may be repeated for five or six days twice daily, a search for and extraction of broken hairs not previously observed being first instituted. This may cut the malady short. If not, Goa powder may be used, which is supposed to be identical with *araroba*, the wood of a tree of the leguminosæ species growing in Brazil, and first brought to India by the Portuguese. A few grains of Goa powder should be mixed with vinegar or limejuice so as to form a paste of the consistency of thick cream, which should be rubbed on with the finger night and morning. Under the action of the Goa powder the part affected becomes whitish, while the surrounding skin is stained brown. Goa powder is also sold under the name *chrysaroline*, and a solution has been prepared which may be applied with a brush. But the real Goa powder is scarce, and often adulterated. It is a fine yellowish powder without smell or taste, and it is as well to see the powder, and not trust to a prepared solution. If the disease is on the face,

care should be taken that the Goa powder does not touch the eyes, as it may cause much irritation. Goa powder has been stated to be an infallible remedy for ringworm of the scalp; but this is not correct, as it frequently fails. Other remedies which may be tried in obstinate cases are *iodine paint*, a solution of carbolic acid ten grains to the ounce, a patent preparation known as *pearl ointment*, and composed of a mixture of lime and lard, which I have known efficacious, and lastly, if all other means fail, the cautious application of mercurial ointment.

But whatever local measures are used, the general health must be attended to. As the fungus grows most luxuriantly on weakly children the diet should be liberal, while cod-liver oil and iron may be given twice a day. When ringworm occurs in schools suspicions may be entertained that the children are underfed and the ventilation imperfect. As ringworm is highly infectious, a child suffering should be isolated as much as possible, and no article of toilet or clothing should be used in common. The healthy should have their heads washed daily with carbolic solution, and frequent search should be instituted on the heads of all, so that any suspected spot may be healed at once.

Tinea, or *ringworm of the body*, is known in the vernacular dialects as *dad*, *dadru*, *majees dad*, *denaii*, also among Europeans as 'dhobees itch,' 'washerwoman's itch,' &c. It commences as a small itching scurfy spot, and enlarging by the circumference shows a line of minute vesicles in more or less luxuriance according to the degree of fungus present, which when copious necessarily excites a greater degree of inflammatory action. It frequently developes about the fork and round the waist, being determined, at least to the latter part, by the irritation of the clothing worn round the body by natives, whose garments are always most plentiful at this part. From the same cause—irritation

— it often becomes more or less eczematous or scaly. Fayrer[1] refers some of these cases to a combination of herpes and tinea, the initiatory patches of *herpes circinnatus* becoming a congenial nidus for the subsequent development of the *trichophyton tonsurans*. From its point of origin it may spread over the body and legs in semicircular patches, the skin getting well after the disease has passed over it. These patches have been called *tinea imbricata*, and have been regarded as a distinct malady, but it is the same in character, although somewhat differing in appearance. Ringworm of the body is attended with considerable itching, especially at night, which keeps the person awake, and tends to destroy the general health, while the scratching induced generates an eczematous or scaly or cracked condition, when it has been mistaken for eczema or psoriasis. It may also attack the chin, when it has been mistaken for sycosis, and it sometimes appears in the roots of the nails.

The *treatment* depends considerably on the seat of the disease, and on the presence or absence of eczematous inflammation. If the parts affected are small, the remedies mentioned for ringworm of the scalp may be used. If a large surface is affected an alkaline wash may be first used, followed by sulphate of zinc ointment (sixty grains to the ounce), iodide of lead ointment (twelve drachms to the ounce), or the *pearl ointment* mentioned at page 494. It must be recollected that in proportion as the general health is improved the more readily is the parasite destroyed by local measures.

PITYRIASIS.—Native name *senwha* or *chulee*. This is perhaps the most common of all Indian skin diseases, prevailing chronically in most parts of the country, and especially in summer, when the affection increases in severity. Pityriasis is much more luxuriant than ever seen in England. The naked backs of boatmen and others are often seen covered

[1] *Med. Times and Gazette*, October, 1874.

with it, and occasionally the whole body is affected. It consists of minute scales, among which a fungus, the *microsporon furfurans*, is said to be detected. These scales fall off with a branny desquamation, and the centre of the patches often fade while the circumference increases. It is attended with little inconvenience, many thinking it not worth notice, but sometimes there is much itching, especially after exertion. The scales although so minute glisten in the sun, which has led to the term *pityriasis versicolor*. Dr. Anthonisez (Columbo) states that when the patches are numerous on the back and chest they give rise to an appearance like tortoiseshell, and are considered a mark of beauty. Erasmus Wilson, however, considered pityrasis as eczema in a very mild form and not caused by the fungus. As regards treatment, there are no better remedies than small doses of arsenic, frequent washing with carbolic acid soap, and attempts to improve the general health.

SCABIES.—Native name *Khujlee*, *Khaj*, *Malabar Khujlee*. The exaggerated scabies which occurs in the East has been described as *scabies ferox*, and is said to be due to an overgrown *acarus scabiei*. But any itch if neglected and irritated will degenerate into conditions presenting appearances very different from the simple itch pustule, or even into open sores. Thus in India as a result of itch the skin is seen thickened, inflamed, greatly crusted, and scabbed from the drying up of discharge from the pustules mixed with acari and their excoriæ. Hence itch is frequently mistaken for other forms of skin disease, as advanced tinea or eczema. I believe, however, that itch very often exists at the same time as one or more other skin diseases, when it is quite impossible to say what the original affection was. The principal diagnostic signs are the discovery of itch pustules on other parts of the body. The sulphur treatment is best. When in charge of the Joudpoor dispensary I frequently had dozens of persons sitting in the yard rubbing each other

with compound sulphur ointment. Cures, however, are comparatively rarely effected, as the natives when relieved do not take the trouble to continue the treatment so as to effect a perfect cure, and they frequently contract new itch.

LICHEN TROPICUS, or 'prickly heat;' native name, *gham-ghur*.—Sangster disposes of lichen tropicus as a temporary lichenous condition preceding the vesicular stage of eczema (Quain's 'Dictionary'), which is not correct. Lichen tropicus is probably the first complaint a new comer to India suffers from, and although unattended with danger it is often very annoying or even distressing. The symptoms are itching, tingling, pricking, and sweating, while the skin is covered with a bright red eruption presenting in the first stage as red papules, which afterwards become watery vesicles or even contain a little matter. In addition to this, there is frequently interspersed the eruption known as *sudamina*, caused by the sweat uplifting the cuticle. Lichen tropicus differs from true lichen in being vesicular, the latter being a papular eruption in which solid lymph-papules are formed and which undergo no further change except absorption and resolution. The anatomical seat of the disorder is said to be in the sweat-follicles, and the itching has been considered to be a consequence of the failure of the sweat function to relieve the skin, the nerves of which are morbidly stimulated by the retained secretion. All this, however, is open to doubt, for an eruption, at first papular, then vesicular, and afterwards in many instances semi-pustular, interspersed with sudamina, will certainly tingle and itch whether in the sweat-follicles or elsewhere. The eruption is increased by anything tending to augment cutaneous circulation, as exercise, warm drinks, and warm clothing. Prickly heat is usually regarded as rather salutary than otherwise, and this view would appear to be supported by the fact that the plethoric and robust are attacked much more frequently

than the anæmic and weak. The immunity which natives enjoy has been attributed to a more simple mode of life and to less clothing, but the mode of life of some 'advanced' natives is scarcely more simple than that of Europeans, and it appears probable some peculiarity of skin not understood, and consequent on their habituation to the climate, is the reason why they do not suffer from prickly heat.

Light clothing, temperate diet, and an occasional aperient are the remedies. As local applications, equal parts of sal volatile and water will be found to allay itching, or two drachms of bicarbonate of potash in half a pint of water. Twenty grains of sulphate of copper dissolved in an ounce of water is much recommended. Rubbing the skin with a rough towel tends to allay the irritation, the heads of the little vesicles or pustules being thus broken, after which they do not itch. Children suffering from prickly heat should be allowed but very little meat, and if thirsty and feverish, citrate of magnesia may be used.

LEUCODERMA, or 'white skin.'—This consists in the development of white patches arising from a deficiency of pigment, but without any textural alteration. It is really a disorder of the pigmentation of the skin depending on an unequal distribution of colouring matter, without tubercles or anæsthesia, and it has no relation to leprosy. The hairs of the affected part are often white, and round the light patch there is often a dark areola. The patches may be of any size and present on any part of the body, giving when numerous a piebald appearance to the surface. When general it constitutes the condition known as *albinism*, the iris being also affected, and the body usually a tawny red. The fairer skinned natives of the north are more liable to the malady than the darker races of the south of India. There is, I believe, no cure for this disease, but I have known amelioration occur from a prolonged course of nitrate of silver.

MORPHŒA.—This disease is, I believe, of frequent occurrence in India, although often confounded with the early eruptive phase of leprosy, or with leucoderma, or with keloid. Morphœa is however distinct from either, and is to be regarded as a fibroid degeneration involving the whole thickness of the skin. As regards leprosy there is this similarity, that in each there is a new deposit which destroys the skin and alters the pigmentation, atrophy following; but in leprosy there is not the white waxy deposit from the outset, and forming the whole disease, but a new deposit of different character, accompanied by evidence of general nutritive disorder, involving especially the nerve-filaments. As regards leucoderma, the distinguishing feature is the total absence of any textural alteration or anæsthesia. As regards keloid, although the skin covering a keloid tumour has a tense shining appearance, and the colour is whitish or pink, it is difficult to imagine how the two could be confused, as keloid is an actual outgrowth of fibro-cellular tissue forming a tumour usually single, unless, as is often the case, developed on cicatrices, when it is usually multiple. Moreover, keloid is not extraordinarily prevalent in India. The confusion appears to have arisen from Dr. Addison describing morphœa under the term keloid, while others have regarded keloid as a form of fibroma.

Morphœa commences as slightly red spots, and when fully formed is characterised by circumscribed patches of various sizes from that of a pea to several inches, looking white or faintly yellow, or dingy, polished, smooth, and non-elevated. The centre of the patches is more or less anæsthetic. At the circumference is a more or less distinct halo of vascularity, and the surrounding skin may be more pigmented than usual. The cuticle shrivels, becoming slightly discoloured or dingy-looking at times, and hence the terms *morphœa alba* and *morphœa nigra*. The back of the neck, the chest, the abdomen, thighs, and arms are the parts

usually affected, but it may appear on the face. When the disease has existed some time, atrophy ensues, the spots becoming thinned by the removal of the wax-like deposits, a scar-like condition resulting (*morphœa atrophica*). Morphœa is believed to be a modified form of *scleroderma*, the former being the localised, the latter the diffused phase of scleriasis (hide-bound disease), or fibroid hypertrophy of the skin. I am unaware of any treatment having the slightest influence on this disease.

FIBROMA. Synonym: *Molluscum; molluscum fibrosum.*—This is a totally distinct disease, not having relationship, as some have thought, with either morphœa, keloid, or leprosy. Although Fox and Farquhar do not regard this as an Eastern malady, Dr. J. Wise states it is common in Bengal, and I have certainly seen many more instances in India in a given time than in England, even when resident surgeon in a large hospital. Moreover, it is well known in Bengal as *meng* or *megh*, while the Mahomedans call it *ghudud* (swelling), and the Hindu physicians *ras-batik* (rheumatic juice); it is also known as *atchali* (warts), and in Rajpootana it is spoken of as *goli-chumree.* It is characterised by the slow growth of numerous more or less pendulous tumours, to the amount sometimes of many hundreds (901 have been counted), and which may attain the size of a goose's egg. They cause no pain, and do not suppurate. Some are pear-shaped and constricted at the base, others more flattened. The skin although it may be stretched is healthy, in this differing from keloid. On some tumours may be observed a small depression communicating with the interior. To the touch they afford different degrees of solidity. On section it is seen they consist of a fibro-cellular envelope containing an atheromatous substance. A contagious variety of molluscum has been described, but this I have not seen. Operative interference has been proposed, either by opening the tumours and taking away the contents

or by removing the tumours altogether. But such measures are manifestly impracticable, unless, as rarely happens, the tumours are very few in number. There is similar ignorance regarding the cause and beneficial medical treatment of this disease.

CHAPTER XXIX.

SNAKE-BITE.

Professor Gmelin, in the *Systema Naturæ*, gives a list of 219 species of snakes, of which one in ten only are poisonous. In Dr. Russell's work on the 'Serpents of India,' forty-three different species are delineated, of which only seven possess poison fangs. Theobald, in his descriptive catalogue of the 'Serpents of India,' gives seventeen species of innocent snakes comprising 180 varieties, and three of poisonous snakes containing thirty-three varieties. Günther made a larger number than other authors named, but he included water snakes, (*Hydrophidæ*) in the total, about which very little is known. There is reason to believe that there are in India 213 known individual species of snakes, of which 33 are poisonous. Thus the comparative number of venomous snakes is less than generally supposed, but it is believed that certain snakes possessing venomous poison-fangs are not mortal to man although they may be destructive to small animals. Of the poisonous Indian snakes there are two well-marked classes, the Colubrine and the Viperine, distinguished by general characteristic appearances. The colubrine snakes are hooded and gradually taper to the tail, while the viperine snakes have triangular-shaped heads and short stumpy tails. In the colubrine snake when dead the skin will be found hanging loose on each side of the neck, and if slight traction be made upon it from both sides simultaneously, the 'spectacles' or *ocellus* characterising the class will be brought into

view. Neither the colubrine nor the viperine snakes have the loreal shield, which is a crescentic-shaped scale directly behind the nasal shield, and found in all innocent snakes (Theobald). The rule is absolute and without exception, that every snake possessing a loreal shield is harmless, although there are some innocuous snakes possessing no loreal (Theobald). The venomous snakes further differ from innocent snakes in the number and arrangement of the teeth. Harmless snakes have two complete rows of ungrooved small teeth, one outer or *maxillary*, and the other inner or *palatine*. In the outer row there are from twenty to twenty-five teeth. But in the venomous snakes this row is represented by one or more poison-fangs only, there being no outer row of small teeth. Fayrer states these fangs are firmly ankylosed to the maxillary bone, which is movable, and that its movement causes the erection or reclination of the fangs. It is believed, however, that in some varieties of snakes the fang itself is movable. In the viperine snakes the fang on one side may be moved independently of the other. The fangs when reclined are covered with a sheath of mucous membrane, in which lay several loose reserve fangs. On opening the mouth a prominent part will be seen on the front portion of the upper jaw immediately before the eyes, which is the poison-fang enveloped as above described. If a piece of stick be pressed against these from behind forwards, the fangs will be raised. In the viperine snake the maxillary bone is reduced in size, but giving insertion to a much larger fang than in the colubrine variety. The fangs also differ in shape and formation. During development the tooth folds on itself like a leaf; but in the *Hydrophidæ* there is an open groove formed; in the cobra there is a complete canal, but in the viper there is a complete tube. In some varieties there is also an opening at the base, so that when the reptile bites the poison is not only carried to the bottom of the puncture inflicted by the point of the fang, but it also escapes by the

opening at the base. When a poison-fang is broken off, a reserve fang gradually grows and takes its place. The poison is secreted by a conglobate racemose gland which represents the parotid of other creatures, and is situated in the temporal region behind the eye. In the cobra it is about the size of an almond, and is furnished with a duct which opens into a capsule of mucous membrane enveloping the base of the fang and communicating with the dental cavity. It is probable that at the orifice of the duct there may be a sphincteral arrangement of muscular fibres, but this has not been demonstrated.

The Indian snakes belong to the order *Ophidia*, of which Fayrer gives three subdivisions, viz., *O. Colubriformis* (innocent), *O. C. Venenosi*, and *O. Viperiformis*. Of the innocent ophidia there are 195 varieties; of the poisonous there are 14 varieties of colubrine and 19 of viperine; grand total of poisonous snakes, 33.

The venomous colubrine snakes ordinarily met with are the 'cobra,' or *naja tripudians*; the *bungarus cæruleus*, or 'krait;' the *ophiophagus elaps*, or 'hamadryad;' the *bungarus fasciatus*, or 'raj-samp.' The viperine snakes commonly met with are the *echis carinata*, or 'phoosa,' and the *daboia Russelli*, or 'chain viper.' Placed in the order of destructiveness (and therefore probably commonness), they stand: 1. Cobra, 2. krait, 3. echis, 4. daboia Russelli, 5. ophiophagus elaps, 6. bungarus fasciatus. A brief description of these snakes is added.

1. *Cobra-de-capello.*—Most common variety *naja tripudians*, but there are several other kinds met with. The usual native name for all varieties is *cobra*, but it is also called *gokarrah* and *karris*. Belongs to the *colubrine* or hooded order. This snake attains to upwards of five feet in length, and six and a quarter inches in girth. It is of various shades of colour, from the darkest olive or black, with a purple iridescence, to pale chocolate, fawn, or yellow, the belly being always much lighter than the upper part. It is further distinguished by the hood, which, expanding when the reptile is excited, discloses the characteristic spectacle-like marks of the class, which marks may be perhaps better described as re-

sembling the 'orbless orbs' of a skull. These marks are light or white rings, with a black or dark centre situated under the hood, and although common to all cobras are most prominent in the dark-coloured snakes. There is also a roundish light mark on the back of the head. The belly is flat, the head short, and not very distant from the body; the nostrils are wide, the eye small, and the pupils round. The scales are smooth, and in numerous series, especially round the hood. The loreal shield is absent. The cobra is a nocturnal snake, comparatively seldom seen in the daytime. It feeds on small animals and insects, and is fond of eggs. It is said to require a good deal of water, and it is able to swim well. It can also climb, and is frequently found in old roofs. It is not of an aggressive nature, and rarely attacks, unless frightened or hurt; but as there are so many cobras, and the poison is so deadly, it still causes the most deaths. When attacking it raises the anterior half of its body, hissing loudly, and with distended hood, the fierce 'death's-head-like' spectacles displayed, eyes glaring, mouth open, and forked tongue protruding, presents a magnificent appearance. When it strikes it slides forward on the posterior half of the body, and thus injures from a longer distance than would be supposed.

2. The *krait*, or *bungarus cæruleus*.—It is known under various native names, the most common being *krait*; in some parts of India it is called *dhomum chili* and *gedi paragoodoo*. It has been known to attain a length of fifty-four inches. There is a single row of hexagonal scales along the centre of the back. The lower part or belly is white or yellowish-white, the upper part bluish or brownish-black, with narrow white streaks not quite so broad as the scales. It has no white streak or collar at the back of the neck, and this serves to distinguish it from other snakes of the order. Next to the cobra it is perhaps the most common and destructive of Indian snakes, and is more aggressive than most others. Like the cobra, it lives in old buildings or ruins, and feeds on small animals and insects. It is fond, especially when small, of creeping beneath carpets or matting, and hence is sometimes spoken of as the 'carpet snake.'

3. *Daboia echis*, or *carinata*; native name, *afae*, or *afai*.—Grows to the length of twenty-five inches. The belly is white, with round brown spots, sometimes so faint as to be scarcely distinguishable. Two white lines run along the body, one on each side, and from these lines stretching transversely across the back there are black marks with central white spots. On the back of the head or crown there is a peculiar white mark, shaped something like a spear's head, and surrounded by a dark or black margin. This snake does not hiss, but makes a noise by the friction of its scales. It is said to be fierce and more aggressive than the cobra, and will frequently attack without cause.

4. *Daboia Russelli*; native names, *ullo boora*, *jessur*, and *siah-chunder*.

This variety of *daboia* is much larger than the *echis*, and quite differently marked. The ground colour is light chocolate, with three series of large black white-edged rings running longitudinally along the body. Those of the middle or central series are rather ovate than circular. The belly is yellowish-brown with darker spots. On each side of the upper surface of the head there is a yellow line, and a triangular brown spot below the eye. This snake is nocturnal in its habits, and not very aggressive.

5. *Ophiophagus elaps*; native names, *shunker chor* (breaker of egg-shells), and *ai raj*.—This snake attains the length of twelve or fourteen feet, and is hooded. The head is short, and there is no loreal shield. The colour is olive green or brown, and the belly is darker than that of any other snake. At intervals of an inch or less, darker wavy streaks run transversely over the back, being much closer together near the neck. The young of this snake are generally much darker in colour than the old. The *ophiophagus elaps* is active and aggressive, and nocturnal in its habits.

6. *Bungarus fasciatus*; native names, *sankni*, *koclia krait*, and *raj samp*.—This snake grows to from four to five feet long, and is easily recognised. The body is trigonal in shape, with a sharp dorsal ridge and declining sides. Transversely across the back there are alternate broad black and yellowish stripes, the margins of which are not wavy as in the last described reptile, but straight. The lower parts, and especially the throat, are more or less yellow. The head is black anteriorly, and is bounded by a yellow V-shaped mark at the neck.

SNAKE-VENOM.—Sufficient venom may be obtained for examination by making the reptile bite through a leaf spread over a cup. During the bite of a full-grown cobra about thirteen grains of poison are expelled. Snake-venom is a colourless liquid of bitter taste, and an average specific gravity of 1658, and often containing bacteria. It has been analysed by Prince Lucien Bonaparte, by Dr. Weir Mitchell, by Dr. De Lacerda, by Reichart and others, and the results of such examinations show that the composition of the venom of different snakes is not the same; also that the poisonous properties do not depend on one special agent. Mitchell and Reichart have succeeded in isolating three distinct proteid bodies, which they have named venom peptone, venom globulin, and venom albumen. The characteristic

effect of the last has not been determined, but that of the two former has been more clearly defined. Venom peptone in particular has been proved to be a powerful putrefacient, and it is not improbable that the difference of symptoms arising from the bite of different snakes may be ultimately referred to the difference of constitution of their poisons. However this may be, we know at present that the poison is deadly to everything cold-blooded or warm-blooded having life, excepting to venomous snakes. According to Milne-Edwards and others, the poison of serpents, including the cobra, is harmless when swallowed, and hence it has been suggested that the salivary, or gastric, or pancreatic ferments might be antidotal. Fayrer, however, states, as the result of experiment, that it may not be applied to either serous or mucous membrane without danger. The fact of permanganate of potash destroying the vitality of snake-venom *in a test-tube* has been proved by Wall and Richards.

Symptoms of Snake-bite.—The bites of poisonous snakes generally show two marks. When there are more than two marks it may be generally safely assumed that the reptile was not poisonous, or that the bites were not inflicted by the poison-fangs. Wall, however, does not place much confidence in the appearance of the marks, as the fangs may not penetrate, but venom flowing from the angle of the mouth may be introduced by wounds inflicted by other teeth. Still the appearance of the wounds as above is, in my experience, characteristic. The parts of the body most frequently bitten are the fingers, toes, ankles, hands, legs, or arms, and often the accident happens while the person is asleep, from which he is aroused by the stinging pain, although not very severe at first. The effects which next take place vary according to the amount and potency of the venom introduced, and according to the variety of snake inflicting the bite. Wall[1] has carefully noted and summarised the differences between

[1] *Indian Snake Poisons*, 1885.

colubrine and viperine poisoning, and from his experiments it would appear that while colubrine poison is a *nerve* poison having a primary effect on the nerves, especially of respiration, leading to carbonic acid poisoning, viperine poison is a *blood* poison and does not effect the respiration so soon. Wall gives other points of distinction afterwards noted; but practically, owing to medical aid being rarely on the spot when a snake-bite takes place, the correspondence of the symptoms in the human subject with those noticed in animals has not been proved. So far as is known, after the pain of the bite, whatever may be the variety of snake inflicting it, faintness, nausea, and perhaps vomiting are the next immediate effects. Then the breathing becomes short and laboured, the pulse quick but intermittent, the powers of speech and swallowing are diminished or even lost, and frothy saliva issues from the mouth, the patient becoming unable to speak or clear the throat. According to Wall, the affection of respiration and the powers of speech takes place quicker in colubrine poisoning than in viperine poisoning. Muscular twitchings next occur, which quickly develope into more or less decided paralysis of the limbs. In the meantime the pain from the wound increases in intensity, and extends upwards towards the body, the absorbent vessels becoming inflamed and appearing on a fair skin as red lines stretching from the wounded part (if on the limbs) toward the groin or armpit. When these effects present early, without much affection of the breathing or flow of saliva, the probability is that the bite is that of a viperine snake, and this probability is increased if convulsions occur, for it appears that the latter poison has no special affinity to particular nerves, but at a later period affects the general nervous system, the respiratory nervous tract being little affected at first. In cobra poisoning the pupil is little affected in the earlier stages, but in viperine poisoning it is widely dilated. Cold sweats, general paralysis, or convulsions now succeed, and

the patient, becoming insensible, sinks sometimes in a few hours. Death may take place from the action of the poison on the cerebral and spinal centres, especially on the medulla, inducing respiratory and general paralysis, or by tetanic arrest of cardiac action, or by a combination of such influences. More commonly, however, especially in viperine poisoning, the case is prolonged several days, and blood-poisoning of a secondary character occurs. The wound becomes discoloured, the limb swells, blisters may form near the injured part, abscesses may occur in any part of the limb; the glands of the groin or axilla, according to the limb injured, may enlarge and suppurate, and the whole surface may be jaundiced. It was long since noticed that such results were apt to follow the bite of the *phoosa* snake (a viper), which is very common in the Bombay Presidency, but no explanation was offered, such as Wall has attempted, based on a difference of the constitution of the venom. Sometimes there is diarrhœa, at others hæmorrhage, from the bowels, nose, or gums; or bleeding takes place from the bite, or from scarifications made in the neighbourhood. There may also be albuminous or bloody urine.

After a snake-bite a large quantity of stimulants are usually given, and when the medical officer arrives he may have to diagnose between the effects of the latter and of the snake-poison.

It is mentioned above that death may occur in a few hours, or the case may be prolonged for days. I have known death take place in less than one hour, and I have known the person linger with erysipelatous inflammation, abscess, and typhoid symptoms for weeks. When death takes place within minutes, fear has probably an influence in the production of syncope. It is on record that a female died from fatal syncope, the result of fear after a bite by an innocent snake. The most fatal period is between the second and third hours.

The viper species, having the largest fangs, make the largest wound and produce the greatest local mischief.[1] Sometimes there is scarcely any mark, excepting the slight punctures ; in other instances, there is a little external swelling. There is, however, distinct change in the parts underneath the skin, from slight hyperæmia of the areola tissue to purple engorgement fading with a pink margin. It may be doubted if this is the effect of the poison as has been surmised, or due to extravasation of blood. Wall, however, believes it to be characteristic, and that if there is no discolouration there is no poisoning.

From cases reported by Russell, Tennant, and other writers, it would appear that snake-venom occasionally induces universal torpor and lethargy without pain. Thus the asp is said to kill in this way, and we are told Cleopatra preferred this painless death. The *Behun* snake of Nepal is also said to induce this result ; and hence has arisen the belief of the country people that the reptile goes abroad during the night, and finding people asleep inhales the breath of its victim until life becomes extinct, the small bite perhaps remaining undetected. This immediate lethargy from snake-bite is certainly uncommon, but inasmuch as instances of the kind have been reported it is well to bear the possibility in mind. If the torpor mentioned by Russell and Gowdie is, as I suspect, a condition of coma immediately occurring, there is a further occasional development of the effects of snake-venom.

Post-mortem appearances differ with respect to the period at which death takes place. When death is early, rigor is well marked and the blood fluid. No microscopical changes in the blood till some hours after death, when the corpuscles change in shape, becoming crenated. Structure of brain usually normal, but the membranes are gorged and the ventricles contain turbid fluid. At a later period of death

[1] Fayrer, *Thanatophidia.*

the parotid glands have been found swollen, the muscles generally of a dirty red colour, with congested lungs and intestines.

Treatment.—It would be an endless task to enumerate all the remedies which have been imposed on credulity as specifics. Notwithstanding the researches of Fayrer, Ewart, V. Richards, Nicholson, Wall, and others, showing there is no such thing as a specific, still specifics are constantly being vaunted in the Indian newspapers. This is unfortunate, as valuable time and lives are often lost while using so-called specifics. Doubtless all vaunting remedies are not charlatans, but err under the confidence of ignorance. Although it has been ascertained that a vigorous cobra can kill several dogs and twenty or thirty fowls before its bite becomes impotent, it does not follow that even when a fresh vigorous cobra bites the injury will be fatal. The poison-fang is of a crooked shape, and it may happen that when the reptile darts on his enemy from an angle the position will not admit the entrance into the skin of the curved point of the venom tooth. In such a case the poison-tooth strikes almost innocuous, as a curved sword would do if it were thrown at a man so that the back of it and not the point should strike him. Yet these are the snake-wounds which look most dangerous. The reptile, feeling it has missed its mark, keeps biting and sawing at the flesh with the fish-like teeth of the palate, and when it lets go its hold blood streams from the wound. Yet the person bitten may not have received the hundredth part of a grain of poison into the system. *Secondly*, it is an ascertained fact that venomous snakes at particular periods of their lives, and at particular periods of the year, especially about the first fall of rain, possess more poison-venom than at other times. *Thirdly*, snakes are more savage if disturbed during the hot part of the day. *Fourthly*, a snake irritated by injury or other causes will secrete more poison-venom. *Fifthly*, the poison

will take greater effect on a person already in a bad state of health, and therefore predisposed to disease. *Sixthly*, there is reason to believe that danger is lessened in correspondence with the bulk of the individual bitten. *Seventhly*, if the bite take place through clothing, much of the poison must be removed by the intervening substance, and less will penetrate the skin; but a fatal case has been recorded where the bite took place through cloth gaiters, Kakee breeches, and drawers (Fayrer). Lastly, the position of the bite has much to do with the danger, as absorption is much more rapid if a vein is injured. In all this, or in the bites of non-venomous snakes, we have an explanation of the apparent recovery of persons from snake-bite when treated by the numerous nostrums at one time or other vaunted as certain remedies. The ancient physicians extolled preparations of the serpent itself. Seneca and Pliny inform us human saliva was believed to be a powerful remedy. But whatever may have been recommended in former times has been equalled in absurdity during the last few years. A native physician, wishing to do me a great service, imparted to me as a certain cure the pulverised skull of a stillborn child. In India the Jogees charm the wound and then give purgatives and opium. Where there are no Jogees the people apply to the wound the root of the *Aristolochia indica* (isurmool) or gogaree wood, ground up and mixed with water. A kind of stone called *zur mora* is also rubbed and placed on the wound. The superstition of killing a white cock and placing the skin of the tail on the wound is prevalent.

Surgeon McCalman, Bombay, has reported [1] cure of the bite of the *echis carinata* by chewing the fresh root of the pangla, an herbaceous shrub growing in the Concan, and described by Dalzell [2] as Pogostemon purpuricaulis (Labiatæ). Information of the effects of this shrub was obtained with

[1] *Bombay Med. and Phys. Soc. Transactions*, 1884.
[2] *Bombay Flora.*

difficulty from a police *patel*, who was observed giving it for snake-bite. It may be hoped further experiment will support what appears to be the successful treatment of one case by pangla.

Although no antidote has yet been discovered for snake-bite, there is no doubt that medical and surgical treatment applied immediately may save the lives of persons thus injured. First, if the injury is anywhere on the limbs (and 94 per cent. of bites do occur on the extremities) tie a bandage or string tightly round the limb three or four inches above the wound, so as to stop the circulation. For this purpose an india-rubber band, if available, is recommended, such as is used in Esmarch's bandage for bloodless operation. The rapidity with which absorption takes place depends a great deal on the part of the body bitten, and on its vascularity. When poison enters a vein it is rapidly fatal, in other instances less so. Wall now advises an incision through the bites, the skin to be reflected on each side for three-fourths of an inch, and the whole of the discoloured areolar tissue to be freely dissected out, especially in the direction of the returning blood-current. If on the ball of the thumb or great toe, he cuts into the muscles; if on the fingers and toes, to the bone; or it may be of advantage to remove one of the less prominent fingers or toes. The dissection is not to be a haphazard cutting away of anything that comes first, but an intelligent and careful dissecting away of the parts holding the poison, bearing in mind the anatomical features of the region. It must also be recollected that the poison may be deep in the areolar tissue, but not directly under the wound, as the skin may have moved before or after the bite. After all has been dissected out Wall advises the wound to be washed with a solution of caustic potash, or of strong permanganate of potash.

Now, in cases coming under my notice I have pinched up the skin and so removed a portion of the latter, as well as

of areolar tissue, but Wall states it is no use pinching up the skin and incising it, as the skin never contains the poison. As, however, I have seen fatal cases of snake-bite when the fang only just penetrated into the skin, and as we know the skin will rapidly absorb other things, I consider it desirable to remove some of the skin surrounding the puncture. Again, after incision, I have been in the habit of encouraging bleeding by if possible immersing the part in hot water, or otherwise by bathing with hot water. Although the ligature stops the circulation some bleeding will still take place, and the escape of blood from the part may be aided by pressure. If no hot water is available apply a 5 per cent. solution of permanganate of potash if available, or some escharotic, as nitric acid, or if not available the actual cautery; taking care that whatever agent is used may penetrate as much as possible in every direction where the poison may seem to have filtrated. After this is done the ligature may be relaxed, for if the virus is destroyed the danger of its entering the circulation is past, and should it already have entered no useful purpose is served by maintaining the ligature. In fact, I have seen a great deal of injury result from keeping a ligature on the limb, as natives often do until not only great œdema results, but blisters occur and mortification threatens. Under any circumstances, when the limb begins to swell and grows a little cold the ligature should be removed.

If the wound is in a position where a ligature cannot be applied, or when a ligature has been applied if the knife cannot be used immediately, I have advised sucking the parts, care being taken that the person performing this office has no sore on the mouth and lips. The great weight of Sir Joseph Fayrer's opinion is against sucking the part, as he believes the snake-venom cannot safely be brought in contact with any mucous membrane. Wall also states that sucking is useless, stating that the attempt to remove a

hypodermic injection of morphia by sucking is useless. Still, as before mentioned, Milne-Edwards and others think snake-venom is not dangerous in contact with unabraded mucous membrane, and in the several instances I have known of a snake-bite sucked immediately after the injury no harm resulted to the person using his mouth, while I believe benefit resulted to the injured person. Surgeon-General Cornish [1] reports a cobra bite on the index finger sucked within ten seconds, followed by ligature, incision, brandy, and recovery, and similar cases may be found scattered through the Indian medical journals. When neither sucking nor knife can be applied, a live coal or stick or red-hot iron may be used. Or if nothing of the kind can be done, a pinch of gunpowder may be placed in the wound and flashed.

The strongest stimulant at hand, whether brandy, whisky, rum, sal volatile, or liquor ammoniæ, should be given at once, and repeated every quarter of an hour. Thirty or 40 drops of liquor ammoniæ may be given, or ounce doses of spirits. To those of less than 15 years of age, 20 drops, and to infants from 3 to 10 of ammonia. The stimulating treatment is not a novel conception, having been mentioned by many authors, medical and lay, especially by Dr. Russell, Boaz, Malte Brun, Tennant, and Forbes, the latter of whom in his 'Oriental Memorandums,' vol. i., states: 'The outward application of eau de luce and a quantity of warm madeira taken internally are generally effectual in curing the bite of the most venomous snake.' Although this cannot be endorsed, the good effects of stimulants administered until the first depressing effects of the poison subside is undoubted. Fayrer insists that the man who is dying from snake-bite is dying from rapid exhaustion of nerve force; therefore anything which excites or disturbs hastens the development of the symptoms. Formerly it

[1] *Ind. Med. Gaz.*, 1880, p. 270.

was advised that if possible the patient should be induced to walk about, and that other means should be employed to combat drowsiness. It is best, however, to keep the patient at rest and so husband his strength. When respiratory failure is prominent, affusion of cold water on the head and face is advisable, and mustard poultices should be applied over the stomach and heart. During the whole treatment the patient should have plenty of fresh air, but at the same time he should be kept moderately warm, especially about the extremities. As soon as the first effects of the poison pass away, the patient should be encouraged to take nourishment in the shape of soup, broth, or Liebig's raw meat tea.

Unfortunately, many cases are not seen until the first effects of the poison have passed away, and the secondary symptoms have more or less developed. But even then the case should not be regarded as necessarily fatal. All that can be done, however, is to give stimulants if necessary, to keep the patient warm and at rest, and if the respiration begins to fail to use cold affusion and artificial respiration. If the absorbents inflame they should be fomented, poultices should be applied to the wound, and to any swelling which may appear about the arm or groin.

Viewing the apparent analogy between curara and snake poisoning, death in both being caused by paralysis of the respiratory apparatus, Brunton and Fayrer hoped that by artificial respiration and support of the bodily temperature they might keep an animal alive until elimination of the poison had taken place; but experiments both by the above named authorities and also by V. Richards failed. Failure may also be stated as the result of every treatment which has been employed, excepting that mentioned in the text, which is occasionally successful.

I reported a case of cobra-bite some years ago, of which the following is a brief *résumé*: Coming home one night, as I blew the candle out,

preparatory to getting into bed, a servant cried out from the verandah he was bitten by a snake. I relighted the candle before the spark of the wick had gone out, and passing the dressing table snatched up a bottle of strong fuming nitric acid which happened to be there, having been obtained the previous day to test some gold. The acid was applied to the snake-bites instantly, a ligature was used, and brandy given. The man remained very depressed for some hours, but made a good recovery. When the snake bit him he hit downwards with his stick, and we found the snake, a large cobra, with a broken back, a few yards away. I regard this man's life as saved from the accident of the nitric acid being at hand.

It should be recollected that venomous snakes will rarely attack if not meddled with. A snake will, as a general rule, retire if it can. And the fact of so many natives being bitten when asleep does not affect the truth of this statement. A snake gliding along passes over the leg, foot, or arm, of the sleeper, when the latter, feeling something, unconsciously moves, and the snake alarmed strikes in self-defence. Similarly, a person is walking through a jungle path, or in the dark, a snake lies in the centre, or at the side, probably asleep. It is disturbed, alarmed, perhaps touched by the passenger, and darts in self-defence at the intruding object. It is, therefore, desirable when proceeding in the dark, in snake-haunted localities, to make as much noise as possible, by treading heavily, or by tapping on the ground with a stick. A little dog about the house is a good protection, and so is a tame mungoose, although this animal is not, as has been supposed, proof against serpent-venom. A line of carbolic acid powder (to which snakes have a great antipathy) in front of a doorway, will prevent a snake passing; but if this protection is used, care must be taken (as I did once) not to prevent a snake already inside proceeding out.

CHAPTER XXX.

SPLEEN DISEASE.

THE spleen lies obliquely in the left hypochondrium, in intimate relation with the diaphragm. In deep expiration its upper end will ascend to the lower edge of the eighth rib, and in deep inspiration it descends as low as the cartilaginous margin of the chest. In health, therefore, the spleen as a general rule is not easily felt. If the stomach contains much food, or the intestines are distended, it is still more difficult to differentiate the spleen. The patient should lie on the right side with the legs flexed, or stand erect. Then percussion should be made somewhat forcibly in a line from the axilla to the ilium. At the ninth or tenth rib the sound becomes dull, denoting the upper boundary of the spleen, which dulness continues to the twelfth rib, where tympanitic sound from the intestines denotes the lower boundary. Horizontally percussion from the median line to a point between the upper and lower boundary, will show where the sound from the hollow stomach gives place to that from the solid viscus. The average size of the spleen is $4\frac{1}{2}$ inches long by 3 broad.

One function of the spleen appears to be the development of white corpuscles, as the blood leaving the organ by the splenic vein is extraordinarily rich in these products. But the red corpuscles being found in the splenic pulp in every stage of disintegration, it has been suggested that the red corpuscles are broken up in the spleen in order to form

pigment, which is then carried to the liver by the splenic vein, to be used in the bile. Some also assume the transition of colourless cells into coloured takes place in the spleen. There is also strong reason for believing that the spleen acts as a storehouse of nutrition during the intervals of feeding, and also as a diverticulum for the circulation during digestion and at other times: uses for which it is fit from its elasticity. Notwithstanding the deleterious effects on the individual from spleen disease, the facts that it has been extirpated, the person living afterwards thirteen years, and that persons live apparently in comfort with enormously enlarged spleens for an indefinite period, prove that it is not absolutely essential to life, and that it may be materially affected without apparent prejudice to health.

Diseases of the spleen are most common in marshy districts and in tropical countries, either as a complication or otherwise of fevers. Splenic affections being so common in the localities named, Dr. Dempster proposed examination of the spleen as the *experimentum crucis* of the malarious nature of a country.

The ordinary tropical affections of the spleen are *acute congestion* or *inflammation*, and *chronic congestion*, both of which may lead to *hypertrophy* and *amyloid* degeneration.

Acute congestion most frequently arises suddenly during the progress of paroxysmal fevers. Whenever the abdominal circulation is embarrassed, and the abdominal veins gorged, as they must be during the cold stage of fever, the spleen in particular becomes distended with blood (or there may even be hæmorrhagic infarctions), and the distension not subsiding at once, the spleen becomes enlarged, although still susceptible of return to its natural bulk on the blood being again removed to the general circulation. Temporary congestion of this nature has indeed appeared to take the place of a renewal of the fever paroxysm, or to be the chief symptom of a very imperfect or masked type of the disease.

In such cases without recognisable permanent enlargement there will be periodical feelings of fulness and distension, aggravated by deep pressure over or under the organ. When the hyperæmic condition is more severe it is attended with oppression in the left hypochondrium, often with considerable pain and tenderness over the organ, the pain sometimes extending to the left shoulders and side. Pain is most acute when the peritoneal covering is involved, and often the patient cannot lie comfortably on the right side. Sometimes there is also pain in the left loin. Less frequently pain has been noted in the left leg, ear, and temple; attributable to the more intimate connection between the spleen and the nerves of the left side of the body. There may also be nausea or vomiting, the matter vomited being occasionally mixed with blood. The stools are sometimes mixed with blood, and epistaxis may occur. If acute congestion occurs unconnected with paroxysmal fever, there is always some heat of skin and a slightly quickened pulse, although often not in any marked degree. Recovery is usually preceded by perspirations, diarrhœa, and copious deposit of lithates in the urine. Acute splenitis is to be distinguished from hepatitis by the position of the pain; from gastritis by sickness not being the cardinal symptom, and by pressure being well borne on the epigastrium; from pneumonia and pleurisy by the absence of stethoscopic sounds; from nephritis from the pain not following the course of the ureter; from peritonitis by its localisation.

Chronic Congestion of the spleen may be a sequel of the acute form, resulting from a repetition of temporary congestion, and perhaps the conversion of coagulated blood into organised tissue; or by the entrance and stagnation of albuminous or fibrinous constituents into the spleen, where they pass through a similar process. But usually chronic enlargement of the spleen comes on so gradually that it is long unattended to, arising without the immediate exciting cause of paroxys-

mal fever, but probably in connection with masked fever (p. 330), generally so slight that the condition is not even noticed. Nothing is more common in malarious districts than finding people who have never suffered from fever with enlarged spleens.

Being in the great majority of instances connected with anæmia, the general condition probably first directs attention to the spleen, which is found more or less increased in size. An enlarged spleen forms a tumour on the left side, passing upwards under the thorax. It is movable, and the sharp anterior margin often presents several more or less well defined notches. A hypertrophied spleen feels tense, but on being cut is found soft, and more or less granular with abundant formation of new lymph-cells. In some cases the organ has been found so enlarged as to weigh from 10 lbs. to 30 lbs., even exceeding the liver in volume. Very large spleens may extend downwards, past the umbilicus and into the right hypochondriac and hypogastric regions, and are not easily mistaken. But smaller spleens require to be diagnosed from enlargement of the left lobe of the liver, which is more to the right than splenic tumour; and from enlargement of the left kidney, which is nearer the spine, more fixed, and if the patient is placed on the hands and knees does not drop down.

Chronic splenitis produces fulness, weight, and pain in the left hypochondrium, extending to the epigastrium, and the whole abdomen in accordance with the size of the organ. When the spleen has not been greatly enlarged abnormal præcordial dulness has been noticed, due to shrinking of the margin of the lung consequent on incomplete expansion, the result of limited respiratory action attending excessive debility. In addition to the physical signs of enlargement, the dull expression of countenance, the pearl-coloured or transparent conjunctivæ, the pale, tremulous, but coated tongue, the blanched lips, the occasional existence of systolic cardiac

murmurs, and the tendency to hæmorrhages evidence the deteriorated anæmic condition established (*vide* anæmia, p. 35). When the spleen is enlarged attacks of partial subacute splenitis are common, and these are usually followed by an increase in the size of the organ and an aggravation of the general symptoms noted above. Occasionally, however, we see spleens of immense size occupying two-thirds of the abdominal cavity and causing little disturbance of health. This is especially the case with children, but the toleration is usually not lasting, and sooner or later the cachectic state described above supervenes.

The various kinds of spleen cachexia into which the malady has been divided are *anæmia splenica*, implying a diminution of the red corpuscles of the blood without increase of white; *leucocytosis*, a diminution of red accompanied by a slight increase of white; *leucæmia*, a great increase of white; *melano-leucæmia*, a collection of pigment in the white corpuscles. But all these are purely arbitrary distinctions, and are merely links in the chain, or phases of the disease (*vide* anæmia, p. 37).

The causes of spleen diseases are usually stated to be malaria, or as Fayrer puts it, 'That is to say, something that operates under certain conditions of heat and moisture, combined with unknown telluric or atmospheric influences, in the presence generally of organic vegetable matter in a state of decay; from which an extraneous and deleterious agent is imbibed into the body, and the entirety of the circulating fluids undergo a potent and morbid alteration. Or it may be that the nervous centres are first affected, and more especially the nervous centres of organic life, and that the organ which is eminently concerned in the evolution of corpuscular germs of the blood may, in a greater degree than any other, be impaired by the poison.' The question, however, is not how the poison acts, but whether there is any poison concerned in the matter. If so, it certainly must be a poison

which operates in a very different manner in different individuals, for while in some cases it produces so-called malarial fever without spleen complication, in other instances it produces spleen affection without malarial fever; at other times simple insidious cachexia without either fever or spleen. The fact is, in hot countries the nutrition of the body becomes seriously depraved under the influence of continued heat and diminished oxygen from rarified air as the primary causes, aided by great alterations of temperature and humidity, by bad feeding, defective personal hygiene, and want of general sanitation; the immediate causes of splenic congestion being anything which destroys the balance of the abdominal circulation, as the cold stage of fevers, or which determines the flow of blood to the abdominal organs, as chill from vicissitudes of temperature. When cold nights succeed hot days, when periodic rains produce a rapid fall of the temperature, when the cool sea-breeze suddenly dispels the hot stagnant morning air, when the frosts of Northern India succeed the furnace-like blasts of the hot weather, sudden transitions of temperature occur, which act with double force on the enfeebled skin of the tropical resident, the consequence being determination of blood to the visceral organs, such repeated actions tending to permanent enlargement and structural alterations of the elastic spleen especially. From remote ages it has been remarked that enlargement of the spleen, and an emaciated condition of the body are often concomitant, and the conclusion has been too hastily drawn that one is the cause of the other; one class regarding the blood deterioration as the origin of the spleen disease, a second class the spleen disease as the origin of the blood-deterioration. It is probable that the blood-deterioration in the nature of anæmia (*vide* p. 16), and the local disorder in the shape of congestion from vicissitudes of temperature or from fever as noted above, proceed *pari passu*. Also, if it be correct that the weight of the spleen increases con-

siderably and contains more blood during the digestive process, there is a periodical natural congestion established, from which the contaminated blood cannot retire without leaving some of its constituents in the shape of fibrinous deposits in that organ. It has also been surmised that atoms of splenic pulp or extravasated blood, changed in their nature and unfit for their proper purposes, might find their way into the circulation through the splenic vein, thus further deteriorating the blood. And if, in addition to the safety valve function, the spleen exerts an influence in common with the lymphatics or the assimilating process, and aids in rendering the crude materials fit to circulate in the blood, we can understand how disease of the organ must react on the system. And if the spleen has further for its office the transformation of white corpuscles into red, we can also imagine how, this action being checked, the white corpuscles undisposed of may pass into the general circulation and their excess become leucocythæmia splenica (*vide* p. 38).

All, however, is to a very large extent conjectural, and the declaration of Hallen still holds good, viz., that as regards the spleen, he was plunging in a region of mere conjecture, darker in the case of this organ than in that of any other viscus.

Sequelæ.—The results of spleen disease are important and varied. There appears to be some similarity between the condition of the blood in scorbutus and chronic splenitis; as in both forms of disease old wounds open afresh, there is a tendency to local suppurations, surgical operations are badly borne, stumps become gangrenous, dropsical effusions occur, and embolism is not unfrequent. In children especially *cancrum oris* is a malady which may be anticipated. Splenic abscess is comparatively rare, but many cases have been recorded, in several attaining an enormous size, even extending to the ilium, or to the liver, and exciting hepatic abscess.[1]

[1] Murray, *Ind. Med. Gaz.*, Jan., 1880.

The *heart* has also been known to become displaced by the encroachment of an enlarged spleen. *Ascites* is another condition found to be not unfrequently associated with disease and enlargement of the spleen; but in very many instances these associations are not cause and effect. Both, indeed, may be consequences of portal obstruction, but I believe ascites following spleen disease without any concomitant hepatic disease is not common. When the spleen has attained a very large size there will always be some amount of fluid in the peritoneum, but this is the result of irritation and pressure, rather than of the diseased spleen *per se*. Affections of the latter organ do not lead to obstructed circulation like those of the liver, which is readily explained by reference to the anatomy of the portal system of blood-vessels. Anything tending to obstruct the passage of blood through the liver would have a more direct effect in inducing effusion than when the spleen alone is in fault. I have occasionally observed ascites occur in malarious or scorbutic cachexia, without any prior accompanying liver or spleen disease. The signs of ascites are not liable to be confounded with other conditions excepting ovarian dropsy, the possibility of which should be borne in mind.

Lastly, there is a morbid condition of the blood known as *melanæmia*, referable to extensive destruction of blood-globules consequent on diseased spleen, with the result of depositions of pigment taking place principally in those organs in which the capillaries are narrowest, as in the kidneys.

Rupture of the Spleen is a danger to which all with large spleens are liable from very slight cause, such as a blow or fall, or even from bodily exertion, often without any external sign of injury. In addition to the enlargement and increased friability of the spleen there are probably adhesions to neighbouring organs, materially lessening the mobility of the gland, all conditions favourable to rupture

without extraordinary violence. McLeod, in his 'Medico-Legal Experiences,' gives several instances of the spleen being purposely ruptured by blows with the naked feet. Heddle,[1] who collected a number of cases of ruptured spleen, remarks that they all occurred in persons who had suffered from malarious fever, or who had resided in malarious localities. Moreover, it has been noted that the majority of instances of ruptured spleen occur during those seasons when the organ is subjected to additional malarious influences. It was mentioned above, that the spleen when enlarged is sometimes ruptured by physical exertion, and of this I met with an instance. A man having a large spleen after riding a camel a long distance suddenly became collapsed, and was brought in dead. The organ was found lacerated. Cases of spontaneous rupture have also been recorded[2] as occurring during the distress and distension of the cold stage of fever. Rupture of the organ is not, however, immediately or even necessarily fatal. A person has been known to live three days after a rupture,[3] and other instances have occurred where laceration of the spleen was diagnosed, but the patient recovered. Also *post-mortem* examinations sometimes disclose cicatrices on the covering, and in the substance of the organ, which could scarcely originate from any other cause than laceration.

The *symptoms* of ruptured spleen are more or less those of collapse, according as the rupture and consequent escape of blood is large or small. Slight ruptures and slight extravasation may cause local inflammatory action, and symptoms of collapse may not present.

The *treatment* of enlarged spleen resolves itself into endeavours to prevent paroxysms of fever; or otherwise, when these have ceased or have not occurred, to renovate the

[1] *Bombay Med. and Phys. Soc. Trans.*, vol. i.
[2] Webb, *Pathologica Indica*, p. 144.
[3] Beatson, 'On Chittagong Fever,' *Ind. An.*, No. 13.

deteriorated blood. Nothing so clearly leads to diminution of volume of the enlarged spleen as measures calculated to improve the condition of the blood.

In *acute congestion* of the spleen, if there is no diarrhœa, the bowels should be acted on by a mixture composed of sulphate of soda, quinine, and sulphate of iron. If there is diarrhœa, the same should be given without sulphate of soda; or, if a more powerful aperient is required, two scruples of pulv. jalap comp. with five grains of ferri sulph. may be given every morning, with iron and quinine three times a day. It should be recollected that in all cases of spleen disease, whether acute or chronic, while a free action of the bowels is very desirable, purgatives should not be given, as formerly, with the view of diminishing the size of the organ. The patient should be kept in bed, and a hot fomentation may be used locally for the relief of pain. If the case is complicated with intermittent or other fever, the relief of the latter must be aimed at as securing the subsidence of the spleen affection. The medicinal treatment of *chronic congestion* or enlargement of the spleen consists in administering tonics as iron and quinine, or, if necessary, the tonic aperients of Twining and Shulbred, which consist of bitartrate of potash, sulphate of iron, and calumba. Other remedies recommended for enlarged spleen are the iodide of lead, administered internally and also employed as an ointment over the organ; the *kala nimmuck*, or coloured salt of the Indian bazaars, consisting chiefly of muriate of soda, with a little sulphur, lime, and a small proportion of oxide of iron; strong sulphuric or strong nitric acid in five-drop doses (the latter recommended); a mixture of aloes, vinegar and garlic, with a portion of *kuzees* or bazaar sulphate of iron (a native remedy), and sulphate of iron given alone. Some years ago Dr. Dickinson (Bengal Service) recommended the *bindaal kernla*, or *luffa echinata*, an indigenous plant of the N. O. *cucurbitaceæ*, as a remedy in spleen disease. The seeds are

said to be powerfully drastic, but an infusion of the stalks is less so. The dose is from one to two ounces of the latter, made by steeping three drachms of the stalks in a pint of water for several hours. This I have used in dispensary practice with apparently good results. Kirk and Webb long since used limejuice with most beneficial effects, and much more recently Evers [1] recommended anti-scorbutic plants generally. In cases where there is any tendency to scurvy (which are numerous) limejuice and anti-scorbutics will be certain to exert a beneficial influence, and they may be used with a sanguine trust in their doing good.

A combination of quinine, iron, and strychnine is often very beneficial, for, as Maclean has observed, the strychnine is an admirable aid to digestion, the quinine exercises its controlling powers over paroxysms of fever, and the iron improves the anæmic condition of the blood. Numerous other medicines have, however, been recommended, such as the hyposulphites (Hill, of Missouri), ergotine by hypodermic administration (Da Costa), arsenic (Mosler), bromide of potassium (R. Williams), nux vomica (Maguire, California), &c., &c. In India, however, the simple treatment as above mentioned is as successful as can be expected. Of external remedies local abstraction of blood and blistering have been lauded. But depletory external treatment equally with depletory internal treatment are contraindicated, the former tending to increase the general cachexia and debility, and the latter in addition being liable to excite dysentery. Puncture of the enlarged spleen with long sharp stylets is a common practice with some native *hukeems*,[2] and Twining recommended the same practice, but of this I have no practical experience, and find no recorded good results. Macnamara[3]

[1] *London Med. Record*, 1885.

[2] The author's 'Native Practice in Rajpootana,' *Ind. An. Med. Sci.*, 1876.

[3] *Ind. An. Med. Sci.*, vol. xv.

some time back stated the biniodide of mercury would be found of great use in hastening the cure of spleen, and that it should be applied over the enlarged organ as described for the cure of goitre. The efficacy of biniodide of mercury ointment in removing chronic splenic enlargement has been endorsed by Maclean and various other writers, but I doubt it, and have seen quite as much good result from the application of the iodine paint. I am, however, sceptical as to the benefit resulting from any external application, for external applications are usually employed in conjunction with other medicines and change of climate, which probably are most instrumental in any relief afforded. Nevertheless, it is desirable to satisfy the patient that all is being done that can be done, and therefore I usually ordered the occasional application of iodine paint. I think wearing a moderately firm bandage will tend to hasten diminution of volume, and this I was in the habit of recommending ; but finding that many patients have no confidence in so simple a remedial agent as pressure, I generally either ordered the iodide paint as above, or the *emplastrum robrans* to be laid over the part and the bandage to be afterwards applied. Warm baths, medicated or otherwise, are useful adjuvants, particularly, according to some authors, Martin's nitro-muriatic acid formula, and especially so if there is any tendency to liver disease. I am not, however, able to endorse the opinions of those who have written decidedly of the efficacy of the nitro-muriatic bath. I have frequently used it, and cannot avoid the conclusion that a simple warm bath is quite as efficacious. It is stated the bath acts more forcibly in hot than cold climates, and that a lax condition of bowels, tending to relieve the spleen, follows its use. These results I have not noticed, and believe the nitro-muriatic acts as any other bath, opening the pores of the skin and promoting elimination. Skovezewsky, believing enlargement of the spleen due to a depressed action

of the vaso-motor nerves, employed 'Faradisation' to diminish the size of the gland; but as the benefits of the electrical treatment were essentially aided by the administration of quinine and other medicines, it is possible the good results reported were as much due to the latter as the former, especially as another continental author asserted reduction of size of the spleen occurs immediately after taking quinine. Unfortunately we do not find this the case in India. There we believe the indication in the treatment of spleen disease is to *combat the cachectic condition*, and as we are unable to remove its cause, this cannot be otherwise fulfilled than by transferring the patient from the influence—that is, by effecting a thorough change of climate.

Children with enlarged spleen should be encouraged to take plenty of milk, with which a little lime-water may be mixed; they should be clothed warmly, the bowels should be kept open by citrate of magnesia, and small doses of sulphate of iron may be given, or if a more powerful aperient is required, Gregory's powder may be employed, and if there are febrile symptoms, citrate of iron and quinine. When European children suffer from enlarged spleen, removal from India is imperatively demanded. If this is impossible, removal from the locality to the sea if practicable, or to the hills if the disease originated near the coast. It will probably be remarked that the use of quinine has not been specially or exclusively recommended for the cure or relief of enlarged spleen. As a tonic, this remedy may exert some slightly beneficial influence, but I do not believe it has any specific effect either on enlarged spleen or so-called malarious cachexia. I cannot call to mind a single instance in which quinine, whether given in large or small doses, produced any appreciable beneficial effect.

In cases of *rupture of the spleen*, no medical treatment is of much utility. Perfect rest and the judicious adminis-

tration of stimulants are indicated; but stimulants must be given with the greatest caution, and only if the pulse is scarcely perceptible, otherwise the excitement of blood-circulation they cause will add to the internal hæmorrhage.

CHAPTER XXXI.

SYPHILIS.

It is not my intention to enter upon a description of this disease, as in essential characteristics it presents little difference to the varieties met with in other parts of the world. The solitary painless sore, which secretes little, becomes indurated, heals without a scar, is rarely phagedænic, but, accompanied by indolent bubo, having a long period of incubation, is frequently met with. But perhaps more frequently it is the multiple painful sore, which secretes much, heals with a scar, is non-indurated, liable to become phagedænic, accompanied by suppurating bubo, having a short period of incubation, which is met with. But still more frequently the sores seen appear to be mixed or composite, partaking of the characters of both the hard and soft sore, yet typical of neither. It has been asserted that in warm climates, syphilis from its commencement runs a more rapid course; but, in my opinion, the influence of climate on primary syphilis is a factor of little importance, although the reverse when secondary symptoms present. There is indeed then an additional gravity attached to such disorders when they occur in the Eastern tropics, inasmuch as they are perhaps the most fertile predisposing causes of endemic disease which can exist in the European system. In the system of the natives it is sufficiently depressing; but in so much as the latter are, if well fed, better able to withstand the general adverse influences of continued heat and a

tropical climate, there is not the same certainty as in the case of the European that the syphilitic-tainted individual will eventually become the subject of that worst form of anæmia, consequent on a combination of the syphilitic, the so-called malarious, and the scorbutic taints. The blood-changes which occur from the syphilitic taint are an increase of albumen and more abundant white corpuscles; a condition allied to that which presents in anæmia. Not only is syphilis thus *per se* so influential in the production of debility and cachexia, but also its reputed specific remedy, although administered with caution, will certainly tend to a considerable extent to prepare the system for the reception and development of malarious and endemic morbific agencies. For mercury also decreases the number of red globules in the blood, and increases the proportion of white. Everyone who has seen much hospital practice, particularly in a large manufacturing city, can have no difficulty in recalling to mind 'the haggard shanks, shrunk eyes, and bony face,' the pallid countenance, the peculiar gait, the sallow colour and loose appearance of integument, characterising the syphilitic, especially when treated, as in days not very remote, with mercury to salivation. If this condition is developed in England, in a bracing and invigorating climate, it cannot be matter of surprise that the heat, malaria, scorbutic taint, and consequent enervation and debility almost inseparable from tropical residence, will render syphilitic cachexia more easily inducible. Thus, the cachexia from the climate, from the disease, from the remedy, and in natives especially from poor living, all combine and re-act in reducing the unhappy patient to that condition of debility which renders him unable to withstand the slightest attack of abdominal disease, such as dysentery and diarrhœa, which not unfrequently closes his earthly career. The medical history sheets of European soldiers show, with almost unvarying and painful regularity, entries such as follows:—'Admitted with primary sore—secondary symptoms—ague

several times—secondaries—ague several times—diarrhœa—dysentery—hepatitis or hepatic abscess—invalided or death.'

I believe the poison of syphilis to be almost as varied in its intensity as the characters of the sore and following results are known to be; and I believe the action of the poison, whatever may be its intensity, differs in various individuals from some inexplicable condition of temperament or constitution. Thus, the most Hunterian-looking chancre, although most liable to be followed by secondaries, may not present such sequel, while other less specific sores are often followed by secondary symptoms. It is generally believed the venereal poison produces its worst effects in scrofulous habits, but I believe the scorbutic taint is quite as powerful a predisposing cause. Yet, apart from either, there appear to be other concealed influences in operation, for it frequently happens that individuals with exactly the same type of primary sore, and with apparently a similar constitution, suffer extremely disproportionate secondary symptoms. I also think that the virus from the same person differs in virulence at various periods, and so will induce varied sores and after symptoms in different constitutions, or even in similar constitutions. Thus I believe I have known the non-indurated or pustulous sore and the Hunterian or indurated chancre arise from the same exposure, the latter being the more likely result, according as the surface is more sensitive, either from constitutional causes, where the ulcer is developed on the integument, or from irritating local causes, in persons whose glans are habitually covered by the prepuce, when the sore occurs beneath that structure, or from both.

In many cases, the commencement of general symptoms is sharply defined by malaise, want of appetite, headache, and a slightly febrile condition. But oftentimes the febrile condition is so marked as to simulate so-called malarious fever, and this either concurrently with an eruption, or apart from any other manifestation. Fourmier, and other

authors, have described an intermittent, remittent, continued, and capricious form of syphilitic fever; the intermittent variety being the most frequent, generally quotidian in its type, simulating to a great degree so-called malarious fever; but the accession always taking place at night, with pains in the bones, and being unaccompanied by any splenic affection. In the other forms, the rise of temperature also always takes place at night, and there is much asthenia; but the dryness of the tongue, sordes of the teeth, delirium, thoracic râles, gurgling, and diarrhœa of typhoid are absent, the tongue remaining clean, the appetite sometimes good, and the bowels being costive. Such syphilitic fevers are said to occur more frequently in men than in women, and I believe that much of the anomalous fever which syphilitic soldiers suffer from in India is rather syphilitic than true ague or other form of so-called malarious fever. I am supported in this view by the vesperine or nocturnal type of these fevers, by the general absence of splenic complications, by the non-occurrence of typhoid symptoms, by accompanying periosteal pains, neuralgia, and other symptoms of syphilis, and by the comparative uselessness of quinine.

Similarly, I believe that a considerable amount of the liver disease from which European soldiers suffer is either purely syphilitic, or greatly aggravated by syphilis. Whenever an anæmic, sallow-complexioned, syphilitic tainted individual complains of anomalous hepatic discomfort or biliary derangement, for which no direct cause can be assigned, the case will probably be connected with syphilitic affection of the liver, especially if tertiary symptoms can be found in other parts of the body. Syphilis, *per se*, is rarely fatal in adults, yet by altering the structure of various organs by deposits of gummatous material, it renders the parts unable to resist accidental congestions or inflammations, and in this manner the liver is especially liable to be affected in India. Thus, as a result partly of syphilis, and partly of climatic

influences, we have subacute intestinal hepatitis followed by atrophy. Also gummy tumours of the liver, which may give rise to anomalous symptoms, arousing suspicion of abscess, and which perhaps may become abscess either by actual conversion into pus, or from the irritation they excite setting up suppurative inflammation (*vide* Liver Disease, p. 432).

Although *gonorrhœa* does not exert the manifest influence towards the induction of debility and cachexia which the constitutional effect of syphilis exerts, still persistent urethral discharge is to be regarded as a powerful predisposing cause of disease. Gonorrhœa having been contracted by individuals, particularly if of scrofulous constitution, a constant more or less gleety discharge, often prostatic, frequently persists for years. This is aggravated by every tendency to anæmia, by every slight attack of fever or other endemic disease, and in its turn again aids climatorial influences in their debilitating action on the system.

Without entering into a discussion of the arguments for and against the mercurial *treatment* of primary or secondary symptoms, I state my belief that the mineral is not necessary for the cure of the mildest form of syphilis, either primary or secondary; and therefore, remembering the urgent necessity of avoiding as much as possible—especially in Indian practice—the exhibition of all mercurials, I consider it is contra-indicated in all primary sores of the non-indurated or simple character. I do not, however, maintain that mercury will not cure this sore; but I submit that the disease will eventually wear itself out and pass away, even although it does so in the shape of mild cutaneous eruptions, sore-throat, &c., and this with far less injury to the constitution than if sooner despatched by the influence of mercury.

When, however, a Hunterian or clearly indurated chancre exists, mercury becomes a remedy of greater necessity, but it must be administered with caution, and only in sufficient

quantity to neutralise the syphilitic poison, otherwise mercurial cachexia will combine with the disease and render the latter state of the patient worse than the first. I have seen cases of destruction of tissue of the mouth and jaw resulting from the mercurial practice of native *hakeems*; the counterparts of which I have read in old European authors. As European medical practice is extended in India doubtless better methods will prevail, as now the case in Europe.

With regard to the treatment of secondaries, I hold that mercury internally is uncalled for and mischievous, and especially is this the case in India. Happily there is another method of effecting all the good which is capable of resulting from mercury. This is the mercurial vapour bath, as so extensively and successfully employed by my late friend Mr. Langston Parker. Having had the charge of this gentleman's syphilitic cases for a term of three years, during my resident surgeoncy at the Queen's Hospital, Birmingham, having paid considerable attention to syphilitic diseases in that institution, where a wide field for the study of the affection existed, and having used the vapour baths in the East, I may be permitted to state the result of my experience, which is, that for secondary symptoms of syphilis occurring in India of whatever variety, there is no remedy so efficient and less costly to the constitution as the mercurial vapour bath has proved to be. Neither is the apparatus required of a costly character: a cane-bottomed chair, a blanket, a pan for the mercurial spirit lamp, and boiling water being all the essential requisites to sublimise the mercury, which may be a couple of drachms of calomel.

The baths may be given every two, three, or four days; diluent drinks are to be administered at the same time to promoted diaphoresis, and during the intervals preparations of iodine may be employed. But it should be recollected, that the iodide of potassium does not cure syphilis, although

temporarily arresting its progress, also that there is reason to believe purpura may be caused by the iodides, especially in debilitated patients. As a general rule, it may be stated that all lowering tendencies aggravate secondary syphilis. Therefore, as sometimes happens when measures as above are not successful, and it may be thought necessary to employ mercury internally, the mildest form should be used, such as a pill containing one-sixth of a grain of iodide of mercury twice daily. There are, however, cases of secondary syphilis, whether of the skin, tongue, or other parts, which resist any and every kind of treatment, especially in India, returning again and again without any evident cause. In such cases change of climate to Europe is advisable, as a means of improving the general health, on the condition of which much may depend.

Respecting the treatment of gonorrhœa I advise, in those few instances in which the malady is brought to notice at the initiative stage, injection every three hours of a weak solution of nitrate of silver (one grain to the ounce) to be repeated for six or eight times. This sometimes cuts the disease short, but if not, or if the malady is not brought to notice at an early stage, I do not advise active interference; rest and aperients with diluent drinks being the better plan. Respecting the treatment of chronic gleet, I am more disposed to place confidence in temperate living, attention to the general health, the use of tonics as iron and quinine, than in either specific medicines or local applications. The daily use of a *very weak* injection of sulphate of zinc is however advisable. If uncertainty exists as to the particular part of the urethra affected, this may probably be discovered by the rough feeling afforded on the passage of a bulbed sound, and the injection may be conveyed to that part by means of a catheter.

Whatever may be advanced against the *lock hospital* system, I regard it as a great safeguard. We have the fact that many years ago, when

the system was abolished in India at the instance of Inspector-General Burke, the amount of venereal disease in the army soon after doubled. And this will again be the case if the arguments—even though apparently enforced by statistics—latterly brought forward by some authorities should result in a second abolition of the lock hospital system. The fact is, the statistics are fallacious. Primary and secondary symptoms are not sufficiently distinguished, and cases contracted in another station are returned as contracted in the places where they developed, or where, at least, they were brought to notice. When in a population such as Bombay, for instance, I see sixty or seventy women suffering from syphilis, taken and placed in a hospital, I hold there must be so many chances less of the spread of disease. Although it is possible disease may sometimes escape detection, and although some females may be discharged while still capable of communicating disease, such conditions do not apply to the great majority who are certified as clean. We should, I think, discarding sentiment, and accepting the inevitable, not only endeavour to maintain the public women clean, but also instruct the soldiery in the importance of cleanliness on their own parts. If there is, as Lustgarten asserts, a specific syphilitic bacillus causing the disease, cleanliness is the best method of destroying such bacillus. If there is no syphilitic bacillus, and it is stated Lustgarten's syphilitic bacillus has been found in other secretions by workers in Cornil's laboratory, then cleanliness is still the best method of getting rid of the discharges which do undoubtedly contain the syphilitic virus, and in which retained discharges there is reason to believe the poison originates. The very fact of the women being subject to periodical examination causes them to maintain themselves in a much more cleanly condition than they otherwise would.

CHAPTER XXXII.

TETANUS.

TETANUS is a disease of antiquity described by Aretæus. Its frequency in India was noticed in 1629 by Bontius, and at the present day it is an undoubted fact that the disease prevails to a greater extent in tropical than in temperate climates. But that it is *extraordinarily* prevalent *throughout* India is not a correct idea. On the contrary, it is comparatively seldom met with except in the large cities. During fifteen years 554 cases were treated in the large Jamsetjee Hospital, Bombay. It may be defined as a disease in which numerous groups of muscles remain in a condition of tonic spasm with paroxysmal exacerbations.

Tetanus as it exists in the East has been subjected to the same distinctions, viz., *idiopathic* and *traumatic*, *eccentric* or *centric*, which is supposed to mark the disease as it occurs in colder climates, and it has been observed that the former is more common in the months of October, November, and December, while the latter was chiefly seen in April, May, and June. Tetanus has also been described as acute and chronic, also as rheumatic, toxic, &c.; and there is the *tetanus neonatorum* of infants. One of the most striking features of mortuary returns from large Indian cities is the high death-rate of tetanus chiefly among infants and children.

Classes most subject.—Coloured races are more liable than Europeans living among them, but the latter are more

liable than in Europe. Males are more subject than females, excepting pregnant women, lying-in women, and those who have aborted. Youth and middle age are more liable than advanced age, although the latter does not confer immunity. The robust and muscular present more examples than the feeble, probably because the former are more exposed to injury and vicissitudes of temperature. The disease is also more prevalent in beaten and retreating armies than among the victorious, probably in a great measure the consequence of the depression from which the former generally suffer.

Varieties.—Traumatic tetanus is a time-honoured variety of the disease, and I believe every author has adopted the distinction. Without, therefore, presuming to deny that tetanic spasm may attend injuries of nervous peripheries, I venture to affirm that other influences have a greater share in the production of tetanus. There is no evidence to prove that so-called traumatic tetanus is the direct result of wounds and injuries. It is stated that the majority of cases occur after lacerated and crushing wounds, with intrusion and presence of foreign bodies, and therefore after gunshot wounds and burns. It is also stated wounds of the extremities are most frequently followed by the disease, and this irrespective of the fact that the limbs are most exposed to injury, and special import has been ascribed to injuries of tendons. It is also stated tetanus may be caused by severe internal injuries, especially of the neck, without external mark; and again many cases of so-called spontaneous tetanus may, it is said, be regarded etiologically speaking as belonging to traumatic tetanus, since in persons affected diseased processes have been found in the interior of the body which have given rise to the state of irritation in the peripheral nerves of internal organs. Thus pleurisy, peritonitis, ulcers of the rectum, equally with the application of ligatures, and surgical operations, especially the operation

of castration, have all been credited as causes. All this might be accepted, especially as we know convulsions frequently occur from the irritation caused by worms or scybalæ in the intestines. But I may be pardoned scepticism when asked to believe that tetanus may occur 'after the most insignificant injuries as the reverse;'[1] that it has been caused by a blow from a schoolmaster's ferule,[2] from the bite on the finger of a tame sparrow,[3] from mere grazing of the heel,[4] or from other injuries only producing abrasion of the cuticle.[5] Neither do I allow that it is 'one of the most mysterious among the numerous consequences of local injury.'[6] In fact, I believe cold, moisture, atmospheric vicissitudes, and particularly transitions from heat to a lower temperature—nowhere more forcibly felt than in India—to be the chief excitants of tetanus. No doubt the malady often follows wounds and injuries, but I believe it to be in the great majority of cases *post hoc* and not *propter*. And I believe it to be accounted for by the change of life, diet, circumstances, exposure, and nervous shock, which an individual severely hurt experiences, aided perhaps by vitiated blood caused by the absorption of a morbid animal poison developed by perverted action of the wounded surface. Ziemssen indeed states the outbreak of tetanus shows a certain analogy to metastatic pyæmia, and Richardson long ago opined that the disease depends on poison developed in a wound by decomposition. Military surgeons have everywhere noticed that the frequency of tetanus among the wounded evidently depends on the influence of temperature. In the records of campaigns, and in the writings of Hennen, Guthrie, Blane, and others, numbers of cases of tetanus are on record occurring to wounded soldiers, but the concomitant

[1] Ziemssen, *Cyclopædia of Medicine.*
[2] Curling Jacksoman, *Essay on Tetanus.*
[3] Watson's *Lectures.*
[4] Elliottson, *Pract. of Med.*
[5] Watson's *Lectures.*
[6] Skey 'On Tetanus,' *Lancet.*

circumstances were cold, moisture, nocturnal air, and exposure, which the observations of Larry particularly show were especially conducive to the origin of the disease. Hennen laid great stress on cold air in motion as an exciting cause. Sir James McGregor states in the Peninsular war, in every disease, and in every stage of wounds, tetanus was a sequence; but he also remarks, in almost similar terms to Sir G. Ballingall, that the affection chiefly occurred in low damp situations 'adjacent to the Nile or near the sea.' Jackson states atmospheric vicissitudes tend much to promote proclivity to the disease, and also mentions that it frequently occurs in India after childbirth, a period when the system is particularly sensitive to external impressions; and we have all probably seen trismus or complete tetanus follow extensive superficial burns, such condition of surface being inordinately sensitive to external impressions from atmospheric changes. Veterinary surgeons know that tetanus often occurs in the horse after clipping, and Mirbeck I think relates the case of a child seized with tetanus as a consequence of ice-cold water thrown on its chest. If the condition of the wound from which tetanus is supposed to arise is regarded, it is often found that the healing process proceeds satisfactorily even to cicatrisation notwithstanding the tetanus, and it is difficult to imagine this taking place in a wound the cause of such a malady. Again, it is asserted that tetanus may occur from a wound after cicatrisation, and this might be admitted so far as cases go in which foreign bodies have been found in the cicatrix. But I confess scepticism as to the theorised irritation of the nervous filaments by contraction of the cicatrix. I am aware that in some instances the nervous twigs leading from a wound have been found congested and softened, but this may be only a result of the general deterioration of the wound surface consequent on that state of system favourable to the tetanic spasm, and not the cause of such phenomena; for if it

were, similar appearances should always be present. Then again, tetanus is often known to prevail in an almost epidemic manner, and this is especially the case in India. During months only isolated instances are met with, during the next few months they are of almost daily occurrence, showing that the nature of the wound has little to do with the origin of the disease. It has been noticed that during the great wars of recent date, such as the American and Franco-German wars, tetanus was much less prevalent than in former campaigns, and this has been attributed to the use of less irritating applications to wounds; but I believe the comparative immunity has arisen from more care being taken of the wounded in recent times, in the matters of cleanliness, superior diet, ventilation and accommodation, than from any other cause. Without denying that tetanic spasms may be caused by eccentric nervous irritation, I believe the wound is more frequently a predisposing cause than an exciting one. All authorities admit that vicissitudes of temperature are one cause of the disease. In fact, a recent author states, 'The outbreak is preceded by an injury, if not by a history of having taken cold,' hence I think it may be justly concluded that traumatic tetanus is referable to the same causes as idiopathic tetanus, wounds only predisposing to the disease by the nervous shock or tainted blood induced.

The scorbutic taint also, I believe, exerts a powerful predisposition, and so does want of ventilation, which may in some degree account for so many natives suffering whose dwellings are always deficient in this essential. Worms, again, are probably excitants. I recollect Laurent of Strasburg asserted, worms were always the excitants even when the disease occurred in wounded men, and other authors have admitted the causation. Here again there may be an explanation of the frequency of tetanus in India, when it occurs in those districts where worms abound (*vide* p. 554), and at those seasons when fruits and vegetables abound,

by which some varieties of worms are introduced into the system.

Symptoms.—Toothache has been noted as a premonitory symptom, but as this so frequently occurs its value is *nil.* The first symptoms generally come on imperceptibly, and are often felt on first awakening from sleep. There is some stiffness of the neck and about the jaws. The patient often supposes this to be due to exposure, and regards it as rheumatic. There may also be difficulty of swallowing, leading to the ejection of fluids through the mouth and nose. Very slight cases may commence and terminate in this manner, or there may be complete trismus which gradually subsides. But generally spasms soon occur in the neck pulling the head backwards, while the jaws are firmly closed. As the disease advances all the muscles of voluntary motion become gradually affected, the countenance exhibits frightful distortion—*risus sardonicus*—and the violent contractions cause opisthotonos (bending of the body forward), emprosthotonos (bending backward), othotonos (when the body is stiff and straight so that the person rests like a lifeless pillar on the heels and head), or pleurothotonos (*tetanus laterales*), when the body is bent on one side. This last form of tetanic spasm has been denied by some authors, but I have certainly witnessed it, although it does not happen so frequently as the other varieties. It is also stated that spasms sometimes commence in the muscles of an injured part, and not about the neck or jaw;[1] also that tetanus is occasionally limited to the wounded side, and Larry went so far as to deduce the position of the subsequent spasms from the situation of the wound. But these characteristics of the disease, if such they are, I have not seen. The pain suffered during the spasmodic attacks is that of an exaggerated cramp in the leg, and the contractions give to the affected muscles a hard board-like feel. Muscles are occasionally

[1] Holmes' *Surgery.*

torn, and teeth and large bones, as of the thigh, have been broken by muscular action. The forearms and hands sometimes escape these spasms. Partial remissions occur at irregular intervals varying from a few minutes to hours, but a permanent rigidity or tonic spasm always remains in proportion with the force, frequency, duration, and extent of the paroxysms, which are generally more frequent during the night than by day. During the intervals the altered state of the countenance and the pallor cause the patient to appear much older. If there is no permanent trismus the tongue may be caught and bitten severely. If the patient sleeps the spasms relax, but generally there is utter insomnia. A painful sensation of pressure at the epigastrium, with pain shooting through to the back, is often complained of, and rolling of the head from side to side has also been noticed. Sometimes proposed voluntary motion gives rise to a paroxysm as in hydrophobia, especially efforts at swallowing, and then the disease has been termed *tetanus hydrophobicus* (Rose). The peripheral impressions which give rise to spasms are frequently insignificant, such as a touch or a prick with a needle, while grosser irritation may not be followed by similar result. It is stated that sometimes previous to the paroxysm an *aura* or impression is felt proceeding from the wound, if any. Motor paralysis is rare, but has been observed in the facial nerves. The pulse is accelerated, especially when the spasms relax, but there is little or no fever, although the temperature rises higher than that of the rectum during the paroxysms. The bowels are generally costive, the urine scanty, acid, depositing lithates, and sometimes containing albumen or sugar, and retention sometimes occurs. The body is usually drenched with perspiration, and sudamina is frequently seen on the face, neck, and chest, and tingling may be felt over the whole surface. Respiration, especially during the paroxysms, is most laborious. With the increase of tonic spasm the constitutional symptoms

become more serious; from the increasing rigidity of the thoracic muscles respiration is more interfered with, and the action of the diaphragm is further hindered by the contraction of the abdominal muscles. One of the great dangers now is spasm of the glottis, which may cause a fatal result so suddenly that, as Dickson wrote, 'While you make your notes that the patient is better, he is seized with spasm, and falls asphyxiated at your feet.' But the danger from dyspnœa has been said to be exaggerated, as the narcotism caused by retained carbonic acid causes a relaxation of spasm. However this may be, urgent dyspnœa, clammy sweat, imperceptible pulse, livid countenance, and perhaps delirium (although consciousness is often undisturbed to the end), indicate a fatal result, which may take place from exhaustion as well as suffocation, either because the system is worn out by the violence of the spasms and the want of nourishment, especially when deglutition is early affected, or because respiration is suspended sufficiently long to cut off all supply of arterialised blood to the brain. Or death may occur from stoppage of the heart (cardiac paralysis). In cases of recovery stiffness and difficulty of moving often remain for some time.

Incubation and duration.—The time between an injury and an outbreak of the disease varies, it is stated, greatly, but the average is from five to ten days. But it may take place in a few hours, or weeks may intervene. Tetanus has sometimes occurred half-an-hour after an operation. The duration of the disease is from two to eight days, but it is said to have proved fatal in a quarter of an hour,[1] and has been prolonged for a month,[2] thus leading to the division of *acute* and *chronic*.

The Hippocratic aphorism, that tetanus ends in recovery if the fourth day is survived, is still worthy of some credence, for recovery is more likely the longer the person survives.

[1] Copeland, *Med. Dict.* [2] Curling Jacksoman, *Essay*.

The prognosis is also more favourable when a long time intervenes between a wound and the attack. A slight degree of trismus is also favourable, as the patient is able to take more nourishment.

Tetanus Neonatorum, or *trismus nascentium*, is a peculiar form of tetanus, and, as previously mentioned, is very common in India. Most cases occur about the second or third week after birth. It is popularly known among Europeans as 'nine-day fit.' It is usually preceded by premonitory symptoms, as restlessness, whimpering, broken sleep, yawning, turning in of the thumbs, and hasty snatches at the mother's nipple, which is soon relinquished. Most probably the first thing which attracts attention is inability of the infant to take the breast properly, which may be erroneously attributed to some fault of the mother's nipple, or tongue-tie of the infant, until at length the child's jaws are noticed to be stiff. Now the muscles of the face become convulsed, the forehead appears wrinkled, the eyelids are spasmodically closed, and the alæ nasi are dilated. The back is curved and stiff, and the other symptoms of tetanus present. The child often dies from suffocation in a few hours. In other instances life may be prolonged with laboured breathing, and the child dies at length from carbonic acid poisoning. Often, owing to the meconium being retained, the surface is yellow. The attack has been attributed to such retention, or to irritation from a neglected navel; but I believe the cause to be exposure to cold.

Post-mortem appearances.—No external change apparent, except an increase of temperature after death. The tetanic spasms of the muscles relax, but are quickly succeeded by *rigor mortis*. The vessels of the brain and spinal cord are full of blood, and there is hyperæmia of the cord itself. Rokitansky found foreign grey substance scattered through the white substance, but this is not constant. Lockhart Clarke found what he regarded as centres of softening in both

grey and white substance. The sympathetic nerve has been found reddened. Changes in internal organs are not constant, but there is usually a vascularity about the mucous membrane of the œsophagus, and cardiac orifice of the stomach; heart often contracted, but has been found in every condition. Blood usually more than ordinarily fluid; muscles pale, with rupture of fibres and extravasated blood; peripheral nerves near a wound sometimes reddened or congested. In some instances abnormal appearances of any kind have not been found. The post-mortem appearances do not add greatly to our knowledge of the nature of the disease, but both symptoms and pathology point to an affection of the nervous centres, which does not appear to be either inflammation or degeneration. In the absence of post-mortem appearances it has been called functional disease of the spinal cord, depending on an excess of what we term motor influence, or in other words an unnatural excitability, or as Todd and Bowman long since designated it 'an increase of spinal polarity,' which was theorised to be aroused to excessive action in some instances at least by an inflammatory or congested condition.

Diagnosis.—The distinction between tetanus and hydrophobia has been indicated under the latter heading (p. 349). The distinction from strychnine poisoning is as follows:—In tetanus some exciting cause, as a wound or a history of exposure, is often present. In poisoning, although the jaws may be firmly closed the mouth can be opened during the intervals between the spasms, and there is no real trismus. Tetanus comes on more gradually than the effects of strychnine, which present in intensity shortly after a poisonous dose has been taken. In tetanus the muscles of mastication and about the neck are first affected, which is rarely the case from poison. In tetanus the spasms do not thoroughly relax even between the paroxysms; in poisoning by strychnine the periodical relaxation is complete. Besides, in poisoning the spasms chiefly affect the extremities; in tetanus the

hands and forearms may escape. In poisoning there are also gastric symptoms not often seen in tetanus. Chronic poisoning by strychnine is more difficult to detect, and is more liable to be mistaken for a chronic form of tetanus than the acute forms.

There is some reason to believe that tetanic conditions sometimes present from nux vomica bark being sold for kurchi bark, or the bark of the Wrightii antidysenterica used much by the natives for abdominal complaints. Cerebro-spinal meningitis may also cause stiff neck, and thus suspicion of tetanus may originate ; but there is no trismus, and the progress of the diseases is distinct (*vide* p. 303).

Treatment.—Numberless have been the remedies used in this disease, and cases of recovery after the most opposite have been recorded, although it certainly cannot be said in consequence of their exhibition. Depletion and depressants have of course been largely used, from the time of Hippocrates downwards. Tartar emetic, bleeding, foxglove, tobacco, &c., have all had their advocates, but experience has, perhaps, demonstrated that less success has followed this treatment than is recorded as the effect of other remedies. Purgatives have been used to an almost incredible extent, such as 280 grains of calomel, with gamboge and jalap in proportion, given in three days. Stimulants have been used to the extent of 110 bottles of port wine, and a cure is recorded by two gallons of brandy.[1] Opium has of course had its advocates, and much testimony is recorded in its favour, perhaps more from the theory that it 'diminishes the irritation of nervous excitement' than from actual good results. In defence of large doses of opium it has been said there is a great toleration, as 'pain eats up the remedy,' but when given in the solid form it has been found undigested in the stomach. Death may also be caused by the coma of narcotics, notwithstanding tetanus being present ; and, moreover, it has been pointed

[1] Holt, *Lancet*, Aug., 1860.

out that associated with narcotism there is a failing action of the heart, and therefore the tendency to death from this cause always existing in tetanus may be aggravated. I believe the best way of using opium in tetanus is inducing the patient to smoke, if he is able to do so, not the comparatively powerless *chandul* used by opium-smokers generally, but the actual drug itself. A successful case from opium-smoking has been recorded by Surgeon-Major Lampry.[1] Some years back sixteen cures out of seventeen cases were reported by the application of ice to the spine, but in after instances this has done no good.[2] Cures have been announced from tincture of aconite, which has signally failed in other instances. The warm bath, the cold bath, and the vapour bath, have each in turn been advocated. Pidduck,[3] of Edinburgh, recommended a special form of vapour bath by which the head is kept always ten or twelve degrees cooler than the body. Cures have also been reported after nicotine, belladonna, atropine, and Indian hemp, first proposed by O'Shaughnessy.[4]

In the abstract of symptoms it is stated if the patient sleeps the muscles relax, and in this there seemed to be an indication of correct practice, and chloroform was naturally selected as the agent. It has not, however, been found so successful as theory would lead us to believe, and was designated by Morehead as treacherous and unsafe, drawing a temporary mask over the symptoms, but allowing the undercurrent to flow with an insidious but certain force till it reaches its crisis, diminishing the volume of the pulse, and causing death to be preceded by low muttering delirium and coma, which are not symptoms of the termination of tetanus uninfluenced by drugs. Chloroform has been tried

[1] *Army Med., San. and Stat. Report*, 1865.
[2] *New York Med. Journal*, Jan. 1860.
[3] *Lancet*, Dec. 15, 1860.
[4] *Brit. and For. Med.-Chirur. Rev.*, 1840.

sufficiently often to show that it is not a cure, although judiciously used it is certainly beneficial.

Worara, or its active principle, curarina, are agents from which much was expected. The former was experimented with years since by Brodie and Morgan.[1] Although cures have been reported from these agents, they have frequently failed both when used internally and hypodermically. Poland truly remarks worara has not at present fulfilled its purport.[2]

In traumatic tetanus it has been recommended that the nerve supplying the injured portion should be divided, but I do not find recorded instances of success after this procedure. If it be admitted that external causes, as atmospheric vicissitudes, are the excitants of the disease, it is evident that this plan is not more worthy of adoption than amputation, formerly so advocated by Larry, and by others, especially Brown-Séquard,[3] since his time. Moreover, it unfortunately happens that the tetanic condition of the spinal cord when established is independent of its local exciting cause if any, and does not cease on its removal.

Experience now assures us that we have no specific remedy for this disease, and that the most we can attempt is to support the strength of the patient, and diminish his sufferings. This is to be fulfilled by quinine wine and nourishment, aided by opium-smoking if practicable, and by chloroform. But the latter must be administered with great caution at lengthened intervals, with a view to moderate anæsthetic effects. Chloral may also be used, and to a greater extent if the chloroform does not seem to agree. When trismus or difficulty of swallowing occurs, most advantage will result from administering remedies and nourishment *per rectum*, rather than removing a tooth for the insertion of a tube, as frequent swallowing is a powerful

[1] Morgan, *Lectures on Tetanus*, 1840.
[2] Holmes' *System of Surgery*. [3] *Ibid.*

excitant of spasm. If a wound co-exist it can do no harm to wash it with a solution of opium, covering it in the meantime with some emollient cataplasm. In all cases quiet and tranquillity should be preserved, with a uniform temperature and the avoidance of any kind of draught or motion of air.

Most of the older writers, as Hennen, Morgan, &c., say they never saw a case of acute tetanus recover, and all allow recovery is the exception. In our present state of knowledge of this disease the practitioner who takes every means of supporting his patient's strength, among which relief of pain is prominent, will be most successful.

The tetanus of infants should be treated in every way as convulsions.

CHAPTER XXXIII.

WORMS.

As in Europe so in India, the three common varieties of worms infesting the human intestines are tape-worms, round-worms, and thread-worms. Among Europeans, tape-worms are most common in adults, round-worms in children, while thread or maw-worms may occur in both children and adults. Among natives tape-worms are most common among flesh-eating Mahomedans, and low caste Hindus whose degraded position allows them to partake of any substance, animal or vegetable, not excepting swine (regarding which we have the Mosaic prohibition) or even animals dying from disease. The Hindoos, who confine themselves to vegetable diet, are most infested with lumbrici, in some parts of the country to the extent, as estimated by certain dispensary returns, of 50 per cent. of the population. Natives of all classes, especially children, suffer from maw-worms. In India, and probably in most warm climates, the system appears predisposed to verminous disease in a remarkable degree. The asthenic habit, the lessened tone of the abdominal viscera, and the frequently impaired digestive powers, lead to morbid accumulations of viscid mucus in the intestines, which form the most fertile soil in which the worms can be reared, whatever may be the primary source from which they proceed. The sources are, however, more abundant in India than in most other countries. The inhabitants of native villages where public latrines have not been established, in common with

the village dogs and pigs, defæcate on the neighbouring fields or waste ground. In the vast pastoral tracts of Western India especially, the cattle and sheep going to pasture in the morning and returning to fold at night, greedily devour the ordure which has been deposited, particularly in the hot weather when grass and fodder is scarce. As a matter of fact, both cattle and sheep eventually become so fond of human ordure as to prefer it to their legitimate food. I have frequently watched cattle waiting for a feast of the kind, become impatient, and butt the biped over to seize the droppings. The researches of Von Siebold of Munich, Kuchenmeister of Zittau, Nelson of Birmingham, and Cobbold of London, leave little reasonable doubt that the embryos of tape-worm may be taken into the stomachs of animals with their food, when, being provided with boring apparatus, they lodge themselves in the flesh, there developing into cysts which, taken into the human stomach in badly cooked meat, grow into tape-worms. Now most of the meat which is consumed in India comes from the grazing districts referred to above; while other meat produced for the market is fed on garbage.

It was shown that out of 13,818 beasts slaughtered in the Punjab in the year 1869, as many as 768 animals were infested with cysticerci. This would be at the rate of about $5\frac{1}{2}$ per cent. It was also shown, on the authority of Dr. James Cleghorn, that of 2,109 beasts slaughtered at Mooltan 899 were diseased, and in most cases both the lungs and liver were affected. This was equivalent to 42 per cent. Or the tape-worm embryo may be eaten with vegetables on which it has been accidentally deposited. Cobbold states the most common parasite known in England was almost entirely nourished by the cellulose and protoplasm of vegetables, and it was by means of a vegetable diet, and by the careless use of unfiltered water employed in the washing of salads and other herbs, that certain parasites were introduced

into the human frame. In India vegetables are certainly not always washed clean, and they are frequently washed with impure water. It is also believed that tape-worm is introduced by using some kinds of fresh-water fish as food, especially the Indian pike or 'singharee.' It is, therefore, evident the sources of tape-worm are most prolific in India.

Lumbricoides, or round-worms, are most prevalent in the coast districts, and it has been supposed they may be introduced into the system by eating fish. Surgeon-General Furnell (Madras) describes a large number of parasites as occurring in fish caught in the eastern seas, among which is the ascaris lumbricoides. But most of the parasites occur in the viscera of the fish, which is not eaten except by the lowest castes, while worms occur in all classes. Moreover, it is considered by Cobbold to be almost certain that the common round-worm completes its life cycle without the necessity of having to pass through the body of any intermediary animal, and it is, therefore, probable that round-worms occur in fish as they do in human beings by the ova being swallowed; which ova it is presumed are generally conveyed into the human stomach at least, through the medium of insufficiently-washed vegetables. Indigenous vegetables or 'turkarree' are more abundant in the moister regions near the sea, becoming less plentiful as the drier interior tracts are reached, thus corresponding with the prevalence of round-worms.

TAPE-WORMS.—The forms most usually met with in India are the *Tænia solium*, and the *Tænia mediocanellata*, the latter being perhaps the most common. The tænia solium is of a white colour and flat, varying in length from five to twenty feet or more. Its shape is uneven, being thick and broader behind. The widest part is about half an inch, tapering anteriorly. The body is jointed and composed of segments, broader than long, each segment fitting into the anterior one. A fully developed worm has some 1,100 of

these joints. Each joint has a male and female organ opening externally. Each worm is therefore a chain of individuals. The head is triangular, about the size of a pin's head, is surrounded by a double row of hooklets, and is further known by four black spots, which are the suckers by which the worm clings to the coats of the bowels. It increases in length by fresh segments developed at the neck, while the fully-formed segments drop off at the tail, full of ova, and pass away with the stools. In the oldest links the ovary is full of eggs, and it is stated the small embryos with their hooklets have been recognised. The cystic representative of the tænia solium is *cysticercus cellulosæ*, most abundantly found in pigs, and known as measly pork.

The *tænia mediocanellata* attains a great length. The links are broader and thicker. The head is furnished with four suckers, but it has no hooklets. Its representative, the *cysticercus*, affects the bovine species.

The *symptoms* of worms vary greatly, some persons being unaware of the presence of worms until attention may be directed to the passage of pieces by stool. The indications commonly present are uneasiness in the bowels, sometimes amounting to pain of a biting or gnawing character. There is frequently irregularity of the bowels, griping pains, straining at stool, fœtid breath, furred tongue, nausea and variable appetite. There is also itching at the nose and fundament. The patient grinds his teeth when asleep, and children often awake frightened and screaming. There may be headache, giddiness, dry cough, palpitation, fainty feeling, and in women hysterical symptoms, or hypochondriacal depression, sometimes leading to suicide (Goopta). Pieces of worm are occasionally passed with the stools, and are the most certain and only conclusive proof of the existence of the parasite; but caution is necessary that pieces of white mucus sometimes passed should not be mistaken for worms. In young children worms sometimes cause progressive emaciation, the

food although taken in fair quantity not seeming to do any good, and the condition may then be mistaken for atrophy. In children worms are also sometimes accompanied by swelling and hardness of the bowels, and they become the exciting cause of convulsions, of infantile remittent fever, and of cholera. Threatenings of paralysis, such as dragging of the limbs, have also been noticed. It frequently occurs that when the symptoms of worms are not well defined, the anomalous feelings present are considered by the patient due to liver complaint, and therefore the possibility of worms existing must always be recollected when obscure symptoms of the kind present. *Anœmia* may be one of the results of tape-worm.

ROUND-WORMS may exist in any part of the intestines and even in the stomach, from which they may be vomited or passed by the mouth. In shape they most resemble the common earth-worm, and are of a pale pink or whitish colour and semi-transparent. There is a circular depression behind the head, and the latter presents three small elevations between which lies the mouth. Each worm contains many thousands of ova. It is recorded[1] that 6,000 round-worms were passed by a child one year and nine months old in five days, 775 in one stool, each worm being from two to seven inches long. The symptoms round-worms cause are very similar to those of tape-worm, but they are more likely to cause griping, purging, diarrhœa, dysenteric symptoms, and especially in children symptoms resembling cholera. In the epidemic cholera of 1883 in Bombay, vomiting of worms was a common accompaniment, showing the number of persons affected in this very wormy locality. But, on the other hand, there may be no symptoms. Sixty-two round-worms were passed in a month, without any worm symptoms.[2] Therefore the alimentary canal may contain an enormous number of these parasites without their producing the usual signs.

[1] *Ind. Med. Gaz.*, April, 1885. [2] *Ibid.*

THREAD-WORMS are about one-third of an inch long, slightly bent, white and semi-transparent. They almost invariably infest the lower part of the bowels near the fundament, where they create much itching and irritation; but it is believed that their head-quarters where they principally breed is much higher, in or about the cæcum. They are not only passed with the fæces, but crawl out during the night in great numbers. They also excite mucous or bloody stools with more or less disturbance of the general health. In females they may crawl into the private parts, causing irritation and discharge. They may also crawl under the foreskin of males with a similar result. Their presence is sometimes attended in children with a milky appearance of the urine. They may also originate protrusion of the bowel. They are most common in weakly dirty children, who may pass hundreds or even thousands of worms. Thread-worms are contracted by swallowing the eggs, of which the worm is full, with food to which the ova have become adherent—or by swallowing fresh germs conveyed to the mouth by the hands of the patient after scratching.

Treatment.—The reason of the success or failure of worm medicines depends much on the manner of taking them. If they reach the worm they kill or at least expel it; if not, they fail. In the case of tape-worm it is particularly necessary they should reach the head of the worm, for although yards of tape-worm may be voided, if the head remains it will grow again, and the old symptoms will return. But the head is exceedingly tenacious of its hold, and is protected by the thick mucus which the irritation of its presence causes the intestines to secrete. It is therefore necessary some preliminary steps should be taken before giving worm medicines. For two days previously, the patient should be put on a light diet of meat, eggs, toasted bread, milk, and green vegetables, avoiding such articles as potatoes, pastry, and farinaceous foods generally. Then on the second night

an aperient should be taken, which may be castor oil for children, and blue pill and colocynth for adults. After these medicines have operated the specific remedy is to be taken. The best remedies for tape-worm are oil of male fern (the basis of Madame Mouffer's famous worm medicine), also called 'liquid extract;' spirits of turpentine (the old remedy of Maddeus), and kossow (first introduced into practice by the late Dr. Vaughan, Bombay Army).

The dose of oil of male fern for an adult is one drachm, one-third part to be given at intervals of half an hour, in some thick gruel, congee, water, or mucilage. Of spirits of turpentine half an ounce, of which half should be given first and the remainder thirty minutes afterwards in some thick fluid. Of powdered kossow half an ounce after steeping for five minutes in a tumbler of hot water, the whole of which should be taken when lukewarm, first well stirring that the powder may be drunk. These remedies should be taken on an empty stomach after preliminary treatment as detailed above, and only liquid food should be allowed for twelve hours, but a dose of castor oil should be taken three or four hours afterwards. Kossow is not recommended for children, the best medicine for a child three or four years old being from twenty to twenty-five minims of oil of male fern, divided into three doses, at intervals of half an hour, as recommended for adults. Or sixty minims of spirits of turpentine may be taken in three divided doses. Or santonin may be used as for round-worms, the precautions regarding liquid diet and a following dose of castor oil being taken as for adults. It is advised that if one remedy fails, the others should be employed in the order named.

For *round-worms* after preceding treatment as for tape-worm, give for an adult five grains of santonin powder at bed-time, the same quantity early next morning, and castor oil one hour afterwards. This failing, turpentine may be used as for tape-worm. For children the best plan is to

give a dose of castor oil the first thing in the morning, and to allow nothing but liquid food during the day. In the evening another dose of castor oil should be administered, and then santonin for a child two or three years old in two-grain doses three times during the next day. While taking the santonin, still only liquid food should be allowed. Santonin combined with calomel is also efficacious.

It should be recollected that peculiar effects have sometimes followed taking santonin. The urine may acquire a reddish tinge, giving rise to suspicion of the presence of blood. Or vision may became affected, every object appearing for a few hours yellow or green. These effects pass off without leaving permanent ill-result. Should santonin fail, oil of male fern and turpentine may be used as recommended for tape-worm.

Other remedies for worms are the *rottlea tinctoria* or *karnala*, an euphorbiaceous plant growing in certain hilly districts in India, and first recommended by McKinnon, of the Bengal Service, but of which I have no experience. A good method of treatment is to give some preparation of iron for a week or ten days, and afterwards a full dose of castor oil. The iron appears to sicken the worm, and thus render it easy of expulsion. Oxide of silver in three-grain doses twice a day, also followed by oil, is sometimes successful. Pomegranate root and bark have been used from the time of Celsus, and their power is not appreciated as it deserves. Decoction of pomegranate may be made by slicing two ounces of fresh root bark and placing it in two pints of water, which is to be boiled down to a pint and strained. Two fluid ounces should be taken fasting, and the dose repeated every half-hour until six draughts have been taken. Nausea or even vomiting may occur after the first dose, but this should not prevent repetition. Then if the bowels are not acted freely upon, a dose of castor oil should be taken.

Besides these named, India contains numerous indigenous

plants which have a greater or less repute among native hukeems as vermifuge remedies. Of these the seeds of the *butea frondosa*, or *palas*; the seeds of the *cucurbita pepo*, or *khondha*; the oil from the pericarp of the *azadarichta Indica* or 'nimb,' and powdered *areca* nut from the *areca catechu*, are the anthelmintics chiefly celebrated as efficient.

Thread-worms are best expelled from adults by giving some saline aperient with iron, and by injecting the rectum daily with twenty grains of quinine in eight ounces of warm water; both infusion of quassia and a table-spoonful of common salt in eight ounces of water are good injections. Castor oil may also be used, which will expel numbers. Children should be given a dose of oil in the evening, and an enema containing quinine or salt the next day after the action of the oil. It is not advisable to give specific remedies for thread-worms which, inhabiting the lower bowels, are not so much exposed to the action of remedies given by the mouth as other kinds of worms; but if measures as above fail, turpentine may be used, and tincture of iron injection employed (two drachms to six ounces of water). It is also desirable to apply carbolic oil round the anus, which will tend to destroy any ova deposited outside. Personal cleanliness is essential, especially avoiding putting the hands to the mouth after application to the anus, which the itching induces.

CHAPTER XXXIV.

WORMS IN THE NOSE.

Synonym :—*Peenash.*

THE affection generally described as 'Worms in the nose,' or *peenash*, is in reality maggots in the nasal passages. It is a malady almost entirely confined to the lower classes of dirty natives, and often the patient will be found to have suffered from some form of syphilitic nasal affection. In some instances, however, the primary condition may be scrofulous or some other affection of the nasal mucous membrane. I never saw a European affected with *peenash*, but a case has been recorded.[1] The disease is caused by a fly entering the nostrils and depositing larvæ which eventually become maggots. Anyone may daily notice flies clustering about the eyes, ears, and nostrils of dirty natives, particularly of children, who take little trouble to rid themselves of the nuisance. At such times, or during sleep or weakness from disease, the flies enter the passage, being often in the first instance attracted by some syphilitic or other nasal discharge. Considerable difference of opinion has been expressed with reference to the variety of fly causing this malady. But the fact is several varieties of fly may cause the disease. Sometimes the maggots produced are nearly half an inch long, of a dirty white colour with ribbed markings, and furnished at the small extremity with a fine double hook of blackish colour. The toughness of their coats, and their almost

[1] *Army Medical Report*, 1870.

absolute immunity for a long time in substances which prove fatal to the larvæ of the ordinary domestic fly, are suggestive of the maggots being of a different species—probably the common blue-bottle. On the other hand, blue-bottles, grey flies, and common house flies have been produced from nasal maggots, some of which do not show the characteristics mentioned above, but are white and softer. In Cayenne, where the malady prevails, it is attributed to the golden fly (*lucilia hominivorax*). Sometimes one or two maggots are passed daily, afterwards several dozens may be passed or extracted. In the advanced stage the disease is one of the most disgusting and loathsome coming under observation. There is constant discharge of offensive pus and occasional epistaxis, while the bridge of the nose gradually becomes depressed, then ulcerated or eaten away, disclosing the interior of the nose filled with a seething coil of maggots. There is also much suffering from the movement of the maggots in the ethmoid cells, and the maggots may also make their way through the palate into the mouth. These cases sometimes terminate rapidly, apparently from meningitis, but most frequently the patient dies worn out by suffering and debility. Maggots are not however confined to the nasal passages, for I have occasionally seen much the same occur in the ear after otitis and perforation of the tympanum. Maggots also are not uncommon in neglected wounds in any part of the body. Peenash is exactly the affection occasionally occurring in the nose of the camel. The piece of wood passing through the septum of the nose, to which the driving string is attached, excites inflammation, discharge ensues, and flies are attracted.

Treatment.—A remedy which will effectually kill the maggots without destroying the structure of their host, has yet to be discovered. I have used solutions of soda, zinc, Condy's fluid, turpentine, lime water, copaibæ, tincture of iron, &c., but I believe the best to be, in the order named, common salt, (two drachms to the ounce), carbolic acid (ten grains to the

ounce), and black wash twice the ordinary strength. Whatever is used should be properly injected three or four times daily, which is not a pleasant operation, and any maggots seen should be picked away with a broad pair of forceps. At the same time every endeavour must be made to support the strength of the patient. I recommend a similar procedure when, as often occurs in India, owing to neglect, wounds become the habitation of maggots. In one case removal of the nose was adopted, but the patient died.[1]

[1] Mayne, *Ind. Med. Gaz.*, Jan., 1875.

CHAPTER XXXV.

UTERINE AFFECTIONS.

Sex is a very important element in forming the statistics of mortality and sickness among Europeans in India. The functions of menstruation, parturition, and lactation exercise in all countries a marked influence on female health, and this influence is most powerful in the tropics. It was stated by one of the most eminent of Anglo-Indian practitioners (Morehead), that 'disease in India is not disease in England,' and the rapidity of the progress of disease in the former country as compared with the latter has been dwelt upon by another, older practitioner (Martin). The truth of the above observations is scarcely illustrated more forcibly than in the maladies of European women. It must be recollected that there are in the female system additional and important organs, especially subject to tropical influences. In the female, to a much greater extent than in the male, the reproductive organs are connected with every vital action. Therefore, there are additional reasons why women in the tropics should, as they often do, break down sooner than men. Many causes combine in inducing a tendency to womb and ovarian diseases in the European female in India. The excitement, novel society, and fatigue consequent on a journey to the tropics, perhaps before the periodic functions have been properly established, is often a first factor. Sea-sickness may also occur, the action of vomiting being sufficient, in some constitutions, to retard, or induce before its time, or to increase the periodic

flux. Then, one of the first effects of a hot tropical climate is a greater tendency to affections of the liver and other abdominal organs, in which condition the womb and its appendages partake. There is also a general relaxation of the tissues, the direct effect of heat, and which seems more decided in the female system than in the male. Sir Joseph Fayrer and Dr. Ewart remark of European females in India, 'The greater confinement to the house, often in dark rooms, and their sedentary lives, with the absence of any regular physical employment, tend to produce laxity of fibre, impairment of digestion and assimilation, and result in a few years in premature ageing, attended by recession of eyes, prominence of the cheeks, and a certain amount of puffiness of features and loss of flesh; or in others, the degeneration assumes the form of obesity, with pastiness and puffiness of the face, and more or less decided pallor.' But I believe the same degeneration may occur simply as a result of heat; as indeed it does occur in soldiers' wives, for instance, who do *not* confine themselves to the house, who do *not* live in darkened rooms, who do *not* lead sedentary lives, to nearly the same extent as the upper classes. Superadded to all this is often habitual exposure during the menstrual period, and neglect of suitable clothing, for it is too frequently believed impossible to take cold in so warm a climate. Then there are errors of diet, leading to intestinal irritation, chills from tatties and punkahs, or after too violent and spasmodic exercise at lawn tennis, or on horseback; the lassitude induced by the heat and the consequent neglect of sufficient moderate regular exercise. Also, probably late hours and a plethora of male society. Even if the individual escapes injury from the influences enumerated above, she probably after a time suffers from attacks of diarrhœa or dysentery, from hæmorrhoids, from intermittent fever, or she miscarries or bears children too quickly. All these causes tend to irritate and weaken the womb, especially dysentery, which

aggravates any womb affection present, and tends to induce it if absent. The greater inability of European females to nurse in India also helps to explain the frequency of womb affection, for the action of suckling promotes the subsidence of the organ to a normal condition after pregnancy, and therefore renders it more liable to be influenced by some of the causes referred to above. It is not, however, European females only who suffer from the diseases incidental to the sex, for native females suffer to a very great extent from one or other of the different forms of uterine disease. This was demonstrated long since by Drs. Tilt and Stewart, the latter of whom, with the authority of his position in the Calcutta Hospital, did not hesitate to say that the majority are habitually subject to deranged menstruation, leucorrhœa, or cervical excoriations. Neither are the reasons difficult to name. Such are the heat of the climate, the early marriages of native females, their sedentary life, and the manner in which native females are treated at their confinement, when they are placed apart in small damp unventilated rooms, and are very liable to puerperal pelvic cellulitis and peritonitis.

Space will not allow a separate consideration of the several disorders of menstruation and of the different diseases of the womb. The most common maladies which arise from the various influences mentioned are at first connected with the monthly discharges, and present as one or other of the varieties of *dysmenorrhœa* and *menorrhagia*. The menstrual flow recurs at irregular intervals, and is frequently attended with great pain, often of a neuralgic, at others of a sub-acute inflammatory character. Frequently the pain is of a very acute, darting character, shooting down the thighs, coming on in severe paroxysms, sometimes so violent as to cause the patient to roll about as if suffering from colic. There may also be nausea, vomiting, diarrhœa, sudden desire to void and pain when passing water. When the pain and tender-

ness in the groins are prominent it indicates that the ovaries are principally implicated (*ovarian dysmenorrhœa*). The patient is also frequently hysterical. Such symptoms may precede the monthly period by a few hours or sometimes days, often twenty-four hours previously is the most painful time. Or the symptoms may appear on the first flow of the discharge; or they may continue with passing of clots of blood, or membranous shreds, until the discharge ceases. Such symptoms may or may not be combined with *menorrhagia* or excessive discharge. As a rule there is most pain when there is least discharge. An immoderate flow in India is generally connected with debility, and is attended with paleness, languor, feeble pulse, and fainty feelings, with dull aching pain in the back, legs, and thighs. Excessive menstruation is very likely to occur in women who have suffered much from over-nursing, or from frequent pregnancy, and sometimes when a doubt of pregnancy exists it may be difficult to distinguish this affection from abortion. Females who suffer thus at the monthly periods are frequently dyspeptic during the intervals, or they may suffer from cough, palpitation, or from pain in the left side, or under the lower part of the left scapula, or in the very lowest part of the spinal column. They are very subject to facial neuralgia, and especially to the pain termed *clavus*. They are also very liable to recurrence of dysmenorrhœa or menorrhagia on slight fatigue, and leucorrhœa is almost always present. The treatment consists in relieving pain when present, which may be accomplished in ovarian dysmenorrhœa by the warm bath, by hot fomentations, and by chloral combined with a stimulant, or by chlorodyne. If hysterical or nervous symptoms are present a tea-spoonful of the following mixture, in water, is often beneficial: Chloroform one drachm, spirits of ammonia aromatic one drachm, brandy one ounce; or if not obtainable, wine or brandy and water will prove temporarily beneficial. But it is not desirable to give either

wine or brandy to these hysterical females if it can be avoided, as recourse to such relief may become habitual, and such agents may be taken when there is no real necessity. Bromide of potassium should be given every second or third hour, and if the pain assumes a periodical character, quinine. When menorrhagia is prominent, the patient should be kept lying down, and everything should be given cold or cool. Stimulants should not be given with the view of combating faintness consequent on the flow, as their action by exciting the circulation tends to increase the discharge, and tendency to syncope will pass away if the patient keeps lying down. Quinine should also be given, not only as an antiperiodic and tonic, but also for the contractile influence which this alkaloid exerts on the womb. During the intervals exercise, short of fatigue, is necessary, but horse exercise should be avoided. A generous but wholesome diet should be adopted, but late hours should be eschewed. The greatest attention should be paid to the ventilation of the sleeping apartment, and when menorrhagia has occurred the bed should be hard and the clothing light. But notwithstanding every care, as time progresses a cachectic condition becomes more and more established, leucorrhœa becomes more excessive from debility and more debilitating from excess, and chronic congestion of the womb, or excoriation, or ulceration of the os uteri result. With increasing debility and cachexia further sub-acute pelvic inflammation and organic disease may be expected, especially if attacks of dysentery recur. Before these conditions present, when dysmenorrhœa or menorrhagia are confirmed in the European female, change of climate ought to be recommended, as the only means by which the sequelæ may be avoided.

There are certain other tropical ailments to which females, and especially European females, are peculiarly liable. It was remarked by Lind that child-bearing was peculiarly fatal to women in Calcutta, and his observation holds good to the

present day. The probability and danger of Bengal fever occurring late in pregnancy has been expatiated upon by various authors (Twining, Martin, Stewart). But such probability and danger are not confined to the fevers seen in Bengal, being common all over the country; the febrile attacks leading to abortion and miscarriage, with all their untoward influences, among which alarming hæmorrhage is prominent. Again, Chevers remarks he has had frequent illustrations that soon after delivery both European and native women often get fever, generally with arrest of the milk and lochia, which fever he terms *malarious post-partum* fever. A similar fever has also been described by Barker, of New York, under the title of *puerperal malarious fever*. It may, however, be doubted whether fevers so occurring are really of a specific nature or anything otherwise than the ordinary milk fevers or puerperal fevers which occur in every country, and which are most liable to present in cachectic subjects, such as the Indian climate produces. Why the female should be so subject to fever before or after parturition has been referred to the womb being an organ especially subject to periodicity, and therefore more likely to be impressed with 'malaria.' But I think there are quite sufficient other causes why the female should be subject to febrile affections at particular times, and that we do not require malaria as an explanation. A similar remark applies to the *pernicious* or intense form of anæmia, from which child-bearing and lying-in women are liable to suffer. Also to the *post-partum hæmorrhage*, so much dreaded in cachectic women in India, that Chevers states it is always desirable to prepare the bed for such a contingency and to give ergot previous to the birth of the child.

Other ailments to which European women are especially liable at particular periods are icterus, hæmorrhoids, tetanus. Harley states the mere normal function of gestation in the human female predisposes to attacks of jaundice much in

the same way as it predisposes to acute yellow atrophy. The latter is comparatively rarely seen in India, but the former is perhaps more liable to occur to females in the tropics than in temperate climates, owing, doubtless, to the general tendency to hepatic disorders in residents in eastern tropical countries.

If it is desirable, as it most certainly is (*vide* p. 11), that European males should not proceed to the tropics before the growth of the body is matured, it is doubly important that females should not so proceed, until not only the growth of the body is matured, but also not until the functions of menstruation are regularly and healthily established. Moreover, those who have suffered from any decided menstrual disorder are advised to remain in a temperate climate. The same remark applies to almost any uterine affection—to that extensive class of maladies women delight in terming 'internal complaints.' Females of chlorotic, anæmic, or sallow appearance should be regarded with suspicion when applicants for advice regarding their suitability for a tropical climate. It may be stated as an axiom that uterine affections will progress from bad to worse in India, that almost any womb affection will be aggravated by the ordinary influence of a tropical climate, the uterine malady itself rendering the person more susceptible to such influence. Hence blood-deterioration, cachexia, neuralgia, and much of the intermittent fever and dysentery from which so many Anglo-Indian females suffer. If women must proceed to or remain in India before the uterine functions are properly established, extraordinary care should be given to the causes of disease which have been sketched.

APPENDIX.

A COMPENDIUM OF DISEASES GENERALLY.

Abdominal Aneurism.—Disease of middle age. *Symptoms.*—Pain of an intermittent neuralgic character in back, abdomen, pelvis, groins, testicles. Sometimes limited to one spot of back, which is tender. Aggravated by movement or stamping; relieved by leaning forwards. Eventually tumour to left of mesial line. Blowing prolonged murmur, heard best in recumbent posture. Fixed local pain in back may be rheumatic; paroxysmal abdominal pain may be due to biliary colic. Disease sometimes simulated by hysteria. May cause jaundice by pressure on bile-ducts; interference with urinary secretion by pressure on renal vessels; vomiting by obstruction of pylorus; displacement of liver forwards, and heart upwards. Towards termination much want of sleep and constitutional irritation. Death usually occurs from rupture. *Treatment.*—Pressure on the proximal side, or on both sides by tourniquets; cures reported, but not generally satisfactory. Horizontal posture, and spare dry diet; cures reported. Galvano-puncture; cures reported. Iodide of potash, large doses; injection of aqueous extract of ergot (ergotine); aconite; cures reported. Palliative, leeches, hypodermic use of morphia.

Albuminuria.—A condition characterised by albumen in the urine. *Symptoms.*—Frequent desire to micturate at night, and the anæmic condition. *Test.*—Boiling urine, and adding nitric acid. Boiling may throw down a cloud of phosphates which disappear under nitric acid; nitric acid may cause a cloud of urates which disappear on boiling; both acid and boiling render albumen visible. Albumen may present from errors of digestion, but is not permanent. In true albuminuria there must be some change

in structure, or circulation of the kidneys. *Treatment.*—For temporary albuminuria, alteration of diet; for permanent, investigation into cause, which may be in the kidneys, or venous congestion of kidneys caused by disease of other organs; counter-irritation over the kidneys, iron.

Alcoholism.—*Cause.*—Habitual drinking. Varieties: 1. *Acute intoxication*, which produces successive and varying phenomena depending on disorder of special and common senses, viz., the friendly, argumentative, captious, communicative, sentimental, quarrelsome, maudlin affectionate, drowsy, and comatose phases. 2. *Acute alcoholic coma*, marked by drowsiness or coma at the first, with stertorous breathing, slow pulse, generally dilated pupils, low temperature. 3. *Chronic alcoholism*, marked by foul breath, jaundiced or injected conjunctivæ, watery eyes, red nose, acne, injected vessels on the cheek, morning cough and expectoration of tough mucus, dyspepsia. The liver is affected in spirit-drinkers by increase of connective tissue, leading to *cirrhosis* and a prematurely aged appearance. In beer-drinkers there is fatty degeneration, and often gouty symptoms. 4. *Delirium tremens*, marked by inability to take food, anxiety, restlessness, tremor of tongue and voluntary muscles, cool skin, frequent profuse perspiration, soft weak pulse, insomnia, hallucinations, attempts at suicide, pupils contracted, eventually typhoid symptoms. In India delirium tremens is frequently aggravated by exposure to the sun. 5. *Alcoholic insanity.* Varieties: *Acute mania, melancholia, chronic dementia, omomania.* In the first there are homicidal impulses; in the second suicidal tendencies; in the third an imbecile condition; in the fourth strange and indecent acts. These patients have generally some hereditary mental defect. *Treatment, for all phases.*—Abstinence from all kinds of alcohol. A *drunken* person is best left alone. Acute coma requires the stomach-pump. In *chronic alcoholism* an easily digested and nourishing diet, with alkalis, effervescent mixtures, capsicum, and hydrocyanic acid if the stomach is irritable. For *insomnia*, bromide and chloral. *Delirium tremens* requires easily assimilated food, such as milk diet, moderate purgation, antimony in one-eighth grain doses if the person is plethoric, bromide or chloral hydrate, good nursing, careful watching, no restraint if possible; if restlessness persists, laudanum; syncope or pneumonia, which sometimes occur, require stimulants.

Angina Pectoris.—Sudden pain, generally on a level with the lower end of the sternum, of a severe stabbing character, but it may extend to the left scapula, shoulder and arm. Sense of faintness or impending death. Face pallid, lips livid, body wet with cold perspiration. May last for minutes or hours, during which the patient fears to breathe. Often comes on during sleep, and may be induced by emotion or physical exertion. Most common after forty years of age. Females least liable. Angina pectoris is often associated with disorders of digestion and of the liver; with gout, albuminuria, diabetes, and certain nervous affections, especially epilepsy; also with organic disease of heart. *Causes.*—Supposed to be irritation of cardiac nerves either directly as in cardiac diseases, or through their connection with the sympathetic and pneumogastric. *Treatment.*—Patient should be allowed to retain the most comfortable position which he naturally takes. Exciting causes should be considered. If the stomach is full of undigested food, an emetic of mustard. If flatulence, peppermint or ether. If attack succeeds exposure to cold, feet in hot mustard and water, and mustard poultice to chest. Five or six minims of nitrite of amyl may be inhaled; nitro-glycerine; stimulants. During intervals endeavour to determine and remove probable cause.

Aorta, Diseases of.—1. *Atheroma*, most common in first portion. Atheromatous matter consists of fat-granules, cholesterine and tissue-débris. Usually middle coat first involved. The atheroma may undergo liquefaction, and projecting the inner coat constitute an *atheromatous abscess.* If this ulcerates a *false aneurism* may result. Or the deposit may undergo calcification by deposit of lime salts. 2. *Fatty degeneration*, usually of the internal coat, which is marbled with minute yellow dots, and the result may be *ulceration* and *false aneurism.* 3. *Co-arctation*, or *stenosis*, in which the aorta is much reduced in calibre from inflammatory thickening or deposits. In such cases the left ventricle becomes dilated and hypertrophied. 4. *Simple dilatation*, without change of structure, usually in the ascending portion, often occurs in connection with renal disease or from hypertrophy of the left ventricle. 5. *Aortitis*, *acute* and *chronic.* Aortitis, acute, is rare, and may result from the irritation of an atheromatous aorta by a thrombus or embolus. Ascending portion usually affected. Morbid changes, hyperæmia, and deposit of fibrine. *Symptoms.*—

Acute substernal pain, palpitation, quick feeble pulse, sometimes harsh systolic murmur. Aortitis, chronic, arises in similar manner; may also arise from, and is always aggravated by labour requiring much muscular exertion, which subjects the aorta to tension from obstruction arising from pressure of contracted muscles on subjacent arteries, and from back pressure of distended veins. As a result the left ventricle becoming hypertrophied, the evils arising from vascular tension are increased. Soldiers are liable to these evils, owing to heavy drill with breathing capacity diminished by faulty dress and accoutrements. 6. *Aneurism.* This is consecutive to disease of the coats as above mentioned, while a definite strain is the immediate cause. Varieties: *True, false, dissecting, varicose. Symptoms.*—Aneurism of the ascending aorta (most common) forms a tumour in second right intercostal space near the sternum. Aneurism of the transverse part is situated at the level of the sternum. Aneurism of the descending aorta, left posterior surface of the thorax near lower dorsal vertebræ. Other symptoms are those arising from excentric pressure, and the physical signs elicited by palpation, percussion, and auscultation. The local pain is dull aching, but there are distant pains arising from pressure on adjacent parts and nerves, in the back, chest, arms, shoulders, abdomen, thighs. When pressure commences to cause absorption of vertebræ there is boring pain in back. But the excentric pressure may cause spasm or paralysis by pressure on the pneumogastric or recurrent laryngeal nerves, evidenced by dysphonia, aphonia, stridor, cough, paroxysmal dyspnœa. Pressure on the pulmonary or cardiac nerves may excite bronchial spasm and angina. Irritation of the cervical sympathetic causes dilatation of the pupil. Pressure on the thoracic duct causes inanition. The tumour formed by aneurism is fixed, soft, and compressible, expanding and contracting with the cardiac pulsation. It varies in the rate and direction of its growth, new symptoms being developed by each encroachment on neighbouring parts. The impulse of the tumour is usually single and systolic in rhythm, but occasionally there is a 'back stroke.' *Fremitus* or thrill is generally present. The murmur is usually single systolic and blowing. Percussion does not afford satisfactory results, as dulness may be due to a tumour of any kind, and dulness behind is too vague; while in front, if tumour is not in contact with thorax, it is modified by intervening organs. Death results suddenly from rupture

and hæmorrhage, or gradually from exhaustion from pain and leakage of blood, for the pressure of the increasing aneurism may cause its own absorption, and consequent leakage. *Treatment.*—Palliative and curative. Pain is relieved by hypodermic injection of morphia, or by chloral; aconite and iodide also of use. Mechanical support when the tumour projects externally. *Curative.*—Absolute rest with regulated diet, use of medicinal agents promoting coagulation within the sac, compression of the artery proximal or distal, distal ligature.

Aphasia.—Defect of speech from cerebral disease. The person understands and thinks, but is incapable of expressing his thoughts. He calls things by wrong names, and in bad cases is unable to arrange his words into a sentence of definite meaning. Most frequently occurs in association with right hemiplegia. Sometimes, while loss of power of speaking correctly is present, power of writing is preserved (*aphemia*). The reverse: *agraphia.* Lesions about the left frontal convolution are most prone to give rise to these conditions. *Aphasia* may follow great excitement or over-work. *Treatment.*—Cessation from work; bromides; patient must be taught to speak again. Treatment of paralysis if present.

Aphthæ.—*Thrush, stomatitis, parasitic stomatitis.* Small white flakes in the mouth, which if detached leave circular ulcers. White patches consist of a microscopic fungus, *oïdium albicans.* May occur in the vagina. Usually presents in debilitated children and old people, and follows improper, insufficient, tainted food, especially soured milk. Attention to these matters, a slight aperient, antacids as lime-water, alum to the ulcers.

Apoplexy.—Prominent features, loss of consciousness without failure of heart's action. Onset often sudden. Face generally flushed, stertorous breathing, pupils usually dilated, or one contracted. In severe cases no reflex action. Patient swallows with difficulty. Escape of fæces, retention of urine. But often premonitory symptoms, such as a partial paralysis, inequality of pupils, deviation of mouth, convulsions, or these may be recognised during the state of coma. Or the malady may commence with headache, or by syncope, which gradually passes into coma. *Cause.*—Cerebral lesion due to anæmia, congestion, thrombosis, embolism, or hæmorrhage. Apoplectic symptoms also result from *uræmia, alcohol,* and *opium.* The uræmic person becomes first drowsy,

then comatose, albumen is present, and there may be pedal œdema. General convulsions at the onset favour uræmia, also persistent depression of temperature. If possible, the retina should be examined for the presence of albuminuric retinitis. *Alcoholic coma* is distinguished by the history of the case, by the smell of liquor in the breath (unreliable, as liquor may have been given to a person with apoplexy); by equally contracted pupils; by the patient being roused, when he babbles incoherently; by any movements which occur being on both sides, while in apoplexy they are limited to one. Struggling is in favour of drink, but general convulsions not so. *Opium coma* is distinguished by the history, by smell of opium in the breath, by equally contracted pupils, by the patient being roused, when he does not babble as in alcoholic coma, by no reflex action from pinching the feet as in apoplexy. Apoplexy is distinguished from *epilepsy* by the stertorous breathing, and in epilepsy there is struggling, the eyes are turned under the lids, and the fit commences with a cry. Late life, absence of spirit or opium smell, profound coma, unequal pupils, sudden onset, local muscular twitching, recognition of paralysis, initial depression of temperature succeeded by rise, are all in favour of cerebral mischief. *Treatment.*—Quiet, recumbent posture, head slightly raised, neck free from constriction, warmth to extremities, cold to head, sinapisms to neck, croton oil. When indication of failure of heart's action careful stimulation. In profound coma in plethoric persons, venesection.

Ascites.—Causes of accumulation within the peritoneum are :—1. *Direct mechanical obstruction*, affecting the portal circulation; 2. *Cardiac* or *pulmonary diseases*, obstructing the general venous circulation; 3. *Disease of kidneys*; 4. *Morbid conditions of the peritoneum*; 5. *Miscellaneous.* Under the *first* head is A. obstruction of the trunk of the portal vein before it enters the liver, from neighbouring tumours or from enlargement of the liver. B. Pressure upon the portal vein within the liver, most usually from cirrhosis. C. Obstruction of the hepatic vein, or inferior vena cava from some neighbouring tumour (rare). Ascites under the *second* head is usually preceded by anasarca of the legs. Under the *third* head ascites is part of a general dropsy. Under the *fourth* head chronic peritonitis (p. 617) is the most frequent cause. Under the *fifth* head there is exposure to cold or wet, suppression of habitual discharges,

rapid cure of cutaneous affections, anæmia, starvation, the scorbutic condition (p. 474). Ascites may be due to combination of causes. The fluid effused is thin, slightly yellow, sometimes more turbid with soft fibrinous flakes. Progress, except in sudden accumulations from cold, is slow. Enlargement general but altering with change of position, being more prominent in the most dependent regions, therefore giving greatest dulness there. Fluctuation. In exceptional cases examination *per vaginam* necessary. With increase of fluid effects of mechanical weight and pressure, as fatigue, flatulence, vomiting, diarrhœa, albuminuria, dyspnœa, palpitations. General symptoms anæmia and wasting. Ascites most likely to be confounded with ovarian tumour. The generally lateral aspect of the latter, and the history of a malady likely to cause ascites are distinctions. *Treatment.*—Discovery of and attention to the condition on which the ascites depends. Absorption of the fluid promoted by hydragogue aperients, diaphoretics, diuretics, baths, and iodide of potassium. Tonics. If no effect produced removal of fluid by aspirator, or trocar and cannula.

Asthma is characterised by severe paroxysmal dyspnœa, often recurring in the night, and due to spasmodic contraction of the bronchi excited by both direct and indirect causes. The former are dust, vegetable irritants, chemical vapours, animal emanations, climatic influences. The latter are nervous, such as emotion, anger, fright (centric), or costive bowels, heavy suppers, flatulence, and other forms of dyspepsia (excitor-motor). Also gout, syphilis, skin diseases, and heredity. A paroxysm of asthma lasts from minutes to days, terminating by cough and expectoration. In the intervals the person is generally well, although in chronic cases a more or less irritable bronchial condition remains. Most common result emphysema, which gives rise to dilatation of the right heart, with prominence of the veins of the breast and neck. Asthma is distinguished from bronchitis by the spasmodic and fugitive character of the dyspnœa, and by the scant expectoration. Aortic aneurism is distinguished by the permanence of the symptoms, and at a later date by the physical sounds and tumour (p. 576). *Treatment* consists in ascertaining the origin of the irritation, and avoidance if possible of the cause. Medicines most useful are stimulants and sedative anti-spasmodics. But what suits one person does no good to another. Chloral hydrate in 15 or 20

grain doses every four hours is most generally beneficial ; smoking stramonium. During the intervals most careful diet.

Atrophy occurs at all periods of life. In infants and children it is due to derangements which interfere with the digestion and elaboration of food. In children up to twelve months old and thereabouts, it may generally be referred to unsuitable food, which sets up gastric catarrh, diarrhœa, or dysentery. Too much farinaceous food is a fertile cause. Between one and three years atrophy is often associated with rickets ; after the age of three with enlarged mesenteric glands or worms. After six with scrofula and phthisis. In adults with phthisis, syphilis, albuminuria, and malarious influences. Atrophy, therefore, can only be combated by treating the maladies of which it is a sign.

Bladder, Diseases of.—The most common are *acute* and *chronic* inflammation. *Acute cystitis* occurs from injuries, foreign bodies as stone, irritants taken internally as cantharides, gonorrhœa. *Symptoms.*—Frequent, painful, urgent micturition ; aching about the pelvis ; pain not relieved by making water ; urine cloudy, perhaps containing pus-cells. Sometimes a false membrane is produced, which females have voided. *Treatment.*—Hip baths, fomentations, recumbent posture, mild diet, no stimulants, liquor potassæ and tincture of henbane, chloral or chlorodyne at night. *Chronic cystitis* generally arises as a consequence of retained urine, from stricture or enlarged prostate (p. 621), but may be a sequel of acute, or result from atony or paralysis. There is frequent desire to make water, the urine is cloudy, and often contains glairy mucus, or pus ; especially that last passed. *Treatment.*—Habitual use of the catheter, injections, buchu, triticum repens, uva ursi, *pareira* brava, alkalis, Vichy and Vals water. The bladder is also liable to *irritability*, marked by frequent desire to urinate, without any evident cause, although it may be due to constipation. To *neuralgia* marked by periodic pain without evident cause, and benefited by quinine or arsenic. To *hypertrophy* and *sacculation*, caused by any obstruction to the flow of urine, chiefly stricture and enlarged prostate. To *paralysis*, which may be temporary from exposure to cold (p. 440), or be part of a more general paralysis from injury or disease of brain or spinal cord. Lastly, to various *morbid growths*, fibrous, villous, vascular, cancerous.

Brain, Diseases of the.—1. *Abscess* may result from encephalitis or injury: origin insidious. *Symptoms.*—Obscure, such as persistent localised headache, twitchings or spasms, partial hemiplegia. Acute pain, vomiting, delirium, dry tongue, are more likely if the abscess is meningeal. *Treatment.*—Trephining, if abscess is diagnosed with confidence; medicines useless. 2. *Anæmia* may be part of a general anæmia, or due to deficient supply of blood, consequent on cardiac weakness. Anatomical characters, pallor of the brain, chiefly in the cortical substance, and few red spots in the white centre. *Symptoms* of general cerebral anæmia, headache, muscæ volitantes, giddiness, buzzing, loss of power of thought, which is better lying down, irritability, insomnia, diminution of muscular power, in severe cases delirium or convulsions. In young children, after exhausting diseases, pallor, somnolence, depressed fontanelle, contracted pupils, diarrhœa, no fever, occasional vomiting, a condition called *spurious hydrocephalus.* Prolonged cerebral anæmia causes permanent damage to brain nutrition, so that the development is arrested in the child, and there is injury to mental power in the adult. Symptoms of sudden anæmia those of syncope, perhaps followed by epileptiform convulsions. *Treatment.*—In sudden anæmia head must be kept low, stimulants, bandages to the limbs. For general anæmia, treatment that of anæmia. 3. *Aneurism.* Aneurism of cerebral arteries is more common than of vessels of similar size elsewhere. The basilar and middle cerebral are most frequently diseased, and the initiative stage is atheromatous degeneration. It is often associated with syphilitic disease or valvular affection. There are few diagnostic symptoms. Intense throbbing, occipital headache occurs in basilar aneurism. When aneurism happens in the internal carotid a murmur has been heard by the patient, and on auscultation. Rupture of an aneurism gives rise to various symptoms, as unilateral paralysis or convulsions, or if blood escapes quickly general paralysis and coma. *Treatment* is vague; if syphilis is suspected, iodide. 4. *Atrophy* may be primary or secondary, general or partial. *Primary* general atrophy is most common in old age, but may follow exhausting diseases, or delirium tremens. *Secondary* atrophy may be general or partial, and may follow various lesions or depend on pressure by a tumour. The anatomical characters are first sub-acute inflammatory condition, then fatty degeneration, then partial absorption, and so atrophy.

Symptoms vague, such as loss of memory, slowness of thought, imbecility. From local atrophy there may be loss of power of a limb, imperfections of speech, strabismus, facial palsy. *Treatment* consists in supporting the patient. 5. *Hydrocephalus*, or *tubercular meningitis*, generally occurs in young scrofulous persons. Depends on tubercular deposits. Often follows eruptive fevers, or is preceded by loss of general health. First *symptom*, frequently a staggering gait, high fever, obstinate vomiting, squinting, aversion to light, alternate flushing and pallor of face, darting paroxysms of pain in the head, peculiar cry (cri hydrocéphalique), hands to the head, rolling of the head, shrunken belly, delirium, boring the fingers into ears or nostrils, convulsions. If bones of head are not united strong pulsation at the fontanelle. Head looks swollen. Hydrocephalus is distinguished from gastric disorder by vomiting continuing after the stomach is empty, by pain of head instead of epigastrium, by the shrunken abdomen, and rolling of the head ; from typhoid with gastric disorder, by the yellow stools, drum-like bowels, and rose eruption of typhoid. *Treatment.*—Darkened room, green blinds, quiet, lancing gums if necessary, change of milk, castor oil, cold to head, mustard poultices to neck, afterwards raw meat soup, stimulants. 6. *Chronic Hydrocephalus ; water on the brain.* Congenital, or may follow acute hydrocephalus ; or may come on gradually. Head often becomes very large, expanding above (hydrocephalic head). Wasting, drowsiness, irritability, diarrhœa, minor convulsive phenomena. Child may live years. Medicine useless. Water has been removed by operation. 7. *Hypertrophy.* A so-called hypertrophied brain is larger and heavier than normal. On removing the skull-cap the encephalon expands. On section a sensation of toughness. Generally developed in rickety children No special symptoms or treatment. 8. *Hyperæmia*, or *congestion*, implies increase of blood in the capillaries of the brain or membranes. *Symptoms.*—Headache, fulness, throbbing, flashes of light, noises in the ears, insomnia, startings, or the signs which sometimes precede apoplexy. *Treatment.*—Leeching, purgation, cold to head, warmth to the extremities. 9. *Inflammation*, or *encephalitis.* Often a sequence of congestion. Some authors profess to distinguish between inflammation of the substance and membranes (*meningitis*), which is not always possible in practice. *Symptoms.*—Shivering, flushes of face, alternating with pallor,

intense burning headache, indicated by cries, or if the patient is a child by application of hands to head, nausea, vomiting, high fever, intolerance of light, sleeplessness, delirium often furious, contracted pupils, abdomen hollow and boat-shaped. Later on slow pulse, dilated pupils, spasms of neck, difficulty of deglutition, paralysis of a limb, convulsions. *Treatment.*—Head should be shaved and ice applied, darkened room, quiet, purgatives, croton oil, mercury. 10. *Softening.* A state of brain tissue, sometimes apparently depending on vascular obstruction, with diminished consistence and fatty degeneration of nerve-fibres. May be blended with sclerosis. Characterised by depression, headache, giddiness, loss of memory, confusion of ideas, imbecility, paralysis. White, red, and yellow softening described. Often follows sunstroke, especially when the person has been a spirit-drinker. Rest from mental work, tonics, if in India removal to a temperate climate. 11. *Sclerosis.* Increase or hypertrophy of the neuroglia, or connective tissue between the nerve cells or fibres, causing increased denseness and compression of nervous substance. If involving brain and cord, called *multiple cerebro-spinal* sclerosis ; if the brain alone, *multiple cerebral sclerosis.* May be circumscribed or diffused, and associated with softening. In infancy it causes imbecility or idiotcy. Multiple cerebral sclerosis is a disease of advanced life, causing pain or trembling of individual or combined muscles of arms, or hands, or other parts, followed by paralysis, or paralysis agitans. 12. *Tumours* have been divided into three series : *first*, those whose centre of origin is one or other of the membranes ; *second*, those springing from blood-vessels ; *third*, those which have the neuroglia as the starting point. Under the *first* class are : A. *Pacchionian granulations* of the arachnoid. B. *Sarcoma*, most usually situated about the sella turcica, consisting of fusiform cells, which may be hard or soft. C. *Myxoma*, a soft, fragile gelatinous growth from the membranes. D. *Psammoma*, usually found in the pineal gland, and consisting of sandy grains, surrounded by connective tissue. E. *Lipoma*, generally from the inner surface of the dura mater, consisting chiefly of fatty matter in cells surrounded by a membrane. Under the *second* head are : A. *Aneurisms*, previously described. B. *Cancer*, generally encephaloid. C. *Fungus* of the dura mater. D. *Tubercle.* Under the third head is : A. *Glioma*, which appears allied to sarcoma. B. *Syphilitic gumma.* C. *Fibroma*,

essentially composed of connective tissue. None of these tumours give rise to definite symptoms, but tumours in *different parts* of the brain may. In the medulla, headache and perhaps convulsions. In the fourth ventricle, sugar in the urine and vomiting. In the cerebellum, occipital headache, irregularity of locomotion, affections of vision, vomiting. In the pons, depression of mind, alterations of speech, disorder of swallowing, hemiplegia. In the pituitary gland, frontal headache and affections of vision. In the middle cavities, the third and fifth nerves are mainly affected. *Treatment* of tumours unsatisfactory. If tumour is diagnosed with syphilitic history, iodide. Beyond this little can be done.

Bright's Disease includes three diseases of the kidneys : 1. *Inflammatory* ; 2. *Waxy*, or *amyloid* ; 3. *Cirrhotic*, or *gouty*. *Inflammatory Bright's disease* is acute or chronic, caused by cold, scarlatinal, and other blood poisons. First there is inflammatory or congested condition ; next fatty degeneration ; then atrophy. *Symptoms.*—Diminution of urine, albuminuria, probably hæmaturia, tube-casts, and dropsy. Death may occur from uræmia. *Waxy* deposit is caused by phthisis, syphilis, and other exhausting conditions, often accompanied by waxy deposits in liver and spleen. Quantity of urine is increased and micturition frequent, urine containing little albumen at first, but afterwards much. No dropsy. Death usually from some intercurrent complication. *Cirrhotic disease* of the kidney occurs chiefly from the abuse of alcohol, or from gout in the system. Consists of an increased growth of the fibrous stroma. Earliest *symptom*, slight albuminuria. After a time the complexion becomes pasty, there is œdema of the conjunctivæ, dyspeptic attacks, and the heart is hypertrophied, and the retina is affected. Death occurs from uræmia. Although pure examples of each of these diseased processes occur, they are frequently combined. The complications of the different forms are gastric affections, characterised by nausea and vomiting ; diarrhœa ; fatty and waxy conditions of the liver ; hypertrophy of the heart, especially in advanced cirrhotic disease ; bronchitis ; anasarca ; gouty affections ; retinitis in the shape of white patches ; uræmic blindness ; uræmia, terminating in convulsions or coma. In Bright's disease, if the quantity of urea passed daily is diminished, as ascertained by the ureameter, uræmia may be expected. *Treatment.*—In the inflammatory form local blood-letting, counter-irritation, diluent drinks, digitalis, hydragogue purgatives,

perchloride of iron, hot-air baths, gallic acid, ergot, nourishing diet. In the treatment of the waxy form the cause of the degeneration should be sought, and removed if possible. For constitutional syphilis, iodide, good food. In the cirrhotic form alcohol is inadmissible, and any gouty tendency should be treated. In the combined forms treatment must be guided by circumstances.

Bronchitis may be acute or chronic. Acute chiefly caused by vicissitudes of temperature ; gout, syphilis, influenza, dentition predisposing. Commences with chilliness, catarrh, sneezing, lachrymation, fulness about nose and eyes, frontal headache, throat dry and sore, hoarseness. Then the affection gradually creeps down the bronchial tubes, and secretion commences. Pain behind the sternum increased by inspiration, tightness of chest, cough at first dry, then with frothy expectoration, afterwards becoming viscid and opaque. When the smaller tubes are attacked (capillary bronchitis) the symptoms are more severe. Temperature reaches 103°. Pulse, 120 to 140. Profuse perspirations. Patient dies of apnœa from fibrinous clots in the heart. Auscultation affords mucous, sub-mucous, and crepitant râles, as the larger or smaller tubes are the seat. Fremitus may be often felt. *Treatment.*—Equable temperature ; free action of the skin should be promoted ; mustard and linseed meal poultices to the chest ; chloral ; opium inadmissible in consequence of tendency to increase the condition of apnœa ; ipecacuanha, squills, iodide if there is a syphilitic taint, colchicum if a gouty taint. *Chronic bronchitis* may result from repeated attacks of acute. Emphysema of the lungs, dilated bronchi, and phthisis cause it. Ordinary chronic bronchitis usually affects elderly people. *Treatment.*—Consider constitutional condition ; attack gout or syphilitic taint. A mild, dry, winter climate, nutritious food, expectorants, wearing a respirator.

Cancrum Oris : *gangrenous stomatitis.*—Commences as dark, hard swelling of cheek, gums, or lips, which ulcerates and sloughs, often involving the bone ; profuse, badly smelling discharge. Occurs to children after exhausting diseases, bad food, and unsanitary conditions. May attack privates of female children, then called *noma*. *Treatment.*—Nitric acid under chloroform, carbolic acid solution, tonics, good food.

Chicken-pox : *varicella* ; *crystalline pock.* — An infectious febrile disease, characterised by successive crops of red pimples,

which in a week pass through the stage of pimple, vesicle, scab. Considered by some to be *modified small-pox*, but the vesicles are not pitted, and do not form matter. *Treatment.*—An aperient and care against cold.

Chorea.—A disease of childhood, most frequent in young girls, characterised by a succession of irregular clonic involuntary movements. A large proportion of children with chorea are found to have had rheumatism; and, whether or not, there is usually a mitral systolic murmur. Poor living, damp lodging, unsanitary surroundings, worms, bad habits, are causes. It has been attributed to capillary embolism in the corpus striatum, but in most fatal cases nothing abnormal is detected. *Treatment.*—Good food, good air, and tonics. When the movements continue during sleep, chloral or morphia, bandaging the limbs.

Convulsions may be *idiopathic* or *symptomatic*; *external* or *internal*; *tonic* or *clonic*. *Predisposing causes.*—An excitable nervous temperament, infancy, mental worry, anxiety, injuries of the head, former attacks of sunstroke. Exciting causes are fright, worms, dentition, an overloaded stomach, constipation, irritation from wounds, tumours and other diseases of the brain and cord, ovarian and uterine irritation, hæmorrhages, tubercular meningitis, the condition of blood in uræmia (p. 633), and in almost any acute disease, especially fevers. *Signs* of convulsions in children are turning in of the thumbs, twitching of face, starting during sleep, night screaming, squinting. When convulsions commence, the arms and legs are drawn inwards and upwards, the eyes turn under the lids, the mouth is awry, the teeth grate, froth appears at the lips, and the head is drawn backwards or sideways; pupils contracted or dilated, insensible to light; discharge of urine or fæces. In bad attacks death from spasmodic closure of the glottis. Otherwise the child cries loudly, or falls asleep, or sinks into a state of stupor, slowly returning to consciousness. *Treatment.*—Fresh air, cold affusion, hot bath, chloroform. If convulsions seem to proceed from overloaded stomach and child can swallow, mustard emetic. If no preceding diarrhœa, half drop of croton oil on back of tongue. Lancing gums if swollen. Afterwards treatment for worms, or any other exciting cause.

Cretinism.—A condition of stunted growth and idiotcy, usually

associated with more or less enlargement of the thyroid gland, met with chiefly in the valleys of mountainous countries. The disease is hereditary, and has been regarded as due to premature ossification of the cranial sutures, especially of the spheno-basilar suture, caused by drinking lime-water by successive generations. *Treatment.*—Removal of the child from the locality, good food, good education, tonics, and cod-liver oil.

Croup.—Often preceded by catarrhal symptoms and hoarseness. Child wakes suddenly with hoarse ringing brassy cough and difficult breathing, the air being drawn in with a hissing metallic sound. These symptoms occur in paroxysms, between which the child sleeps. Cough at first dry, but at length mucus is brought up, and the disease may subside. This, the *first stage* of croup, is inflammatory addition to the state described as spasmodic croup (p. 604). If attack does not terminate as above, the characteristic croupous membrane forms in the trachea and larynx, and tubes or flakes may be coughed up. Efforts to bring up this material cause flushed livid countenance and perspiration. The child sometimes sits, sometimes lies ; the head may be bent backwards, and the hands are frequently applied to the throat ; child may seem better during the day. Often there is amendment after a considerable amount of flaky material has been coughed up ; otherwise death, partly from exhaustion, partly from suffocation, or during convulsions. *Cause* unknown ; low damp positions favourable, especially if exposed to N.E. winds. For distinction between croup and diphtheria *v.* p. 588. *Treatment.*—On first appearance of croupy cough or hoarseness, ipecacuanha and camphorated tincture of opium, and protection from cold, especially at night. Several lamps or a fire may be lighted to increase temperature of bed-room. A teasponful of salad oil given lubricates the throat and decreases the brassy cough. Under such means the attack may pass off as a common cold with running from the nose. When undoubted croup is present, an emetic of ipecac wine and a warm bath. Afterwards small doses of ipecac wine. Emetic and warm bath may be repeated. If a robust child, leeches to upper part of sternum, one for each year of the child's age. Opium and narcotics counterindicated. Atmosphere of room may be rendered moist by steam from boiling water. Object is to combat inflammation, not to weaken the

child, therefore strong broths or other nourishing fluids ; if the child cannot swallow, they should be given as injections. In latter stages stimulants required.

Diphtheria is allied to croup, consisting also in the formation of a membranous substance in and over the parts about the throat. Ordinarily, the first symptoms are depression, chilliness, nausea, stiffness and redness of the throat, difficulty of swallowing. After a variable time, small white specs appear on the inflamed part, which, extending, may cover the whole surface. The diphtheritic process is associated with vegetable organisms (micrococci), which have been regarded as the cause of the disease. The exudation may spread to the mouth, lips, nose, larynx, trachea, conjunctivæ, vagina, rectum, intestines. Glands of neck swollen. Always much fever. Spontaneous separation and discharge of membrane gives much relief. Growing obstruction of breathing and lividity of face and lips point to fatal termination. Hæmorrhage from the throat, or petechiæ on the body, or albumen in the urine unfavourable. There may be very mild or very severe cases, the latter marked by great depression and early typhoid symptoms. Distinctions between croup and diphtheria are the formation of the membrane of diphtheria over the tonsils and in front of the glottis, while in croup it forms inside the larynx, but both may spread upwards or downwards. Diphtheria mostly occurs to adults, croup to children under ten. Diphtheria prevails at all seasons and in all kinds of weather; croup mostly during cold moist weather with N.E. winds. Diphtheria has been considered to be caused, or at least favoured, by bad sewerage, with which croup is not specially connected. Diphtheria is contagious, spreading through a family ; a child with croup does not give it to others, although in the same room. It is also stated the morbid deposit of diphtheria is secreted *in* the substance of the mucous membrane, that of croup *on* the surface. But the similarity in other respects has led to the term *diphtheritic croup*. Diphtheria is distinguished from scarlet fever by the white deposit in the throat, and the absence of rash on the second day. Convalescence may be retarded by albuminuria, pneumonia, paralysis. *Treatment.*— Good ventilation, quiet, rest, removal if there is deficient sewerage, fomentations, chlorate of potash tablets, a ten-grain solution of nitrate of silver to the throat, carbolic acid spray by an atomizer, iron, quinine, chlorate of potassium, good broths, port

wine, digested enemas, tracheotomy. Disinfection or destruction of everything used by the patient; disinfection of hands of attendant, who should avoid inhaling the breath, or their own lips or mouth coming in contact with the expectoration of the patient.

Dentition.—As a rule the first tooth appears at the seventh month, but may appear much earlier. Ordinarily the teeth present as follows: lower central incisors, upper central incisors, upper lateral incisors, lower lateral incisors, first molars, canine, back molars. A child twelve months old should have eight teeth and be cutting first molars. But this order is not invariable. Dentition produces flow of saliva, swelling of gums, flushed cheeks, irritability, restlessness at night, pyrexia. There may be aphthæ, diarrhœa, catarrh, bronchitis, skin eruptions, swelling of glands, startings, twitches, convulsions. Temperature of a teething infant often highest in the morning. *Treatment.*—Lancing gums, if hot and swollen, and the appropriate remedies for complications.

Dropsy.—Accumulation of serous fluid in the subcutaneous cellular tissue, or in a serous cavity. When in the cellular tissue, called *œdema* or *anasarca*; in the peritoneal cavity, *ascites*; in other cavities, *hydropericardium*, *hydrocephalus*, *hydrocele*, *hydrops articuli*, *hydrothorax*. General dropsy is usually the result of albuminuria depending on fatty degeneration of the kidneys. The next most common cause of dropsy is tricuspid regurgitation obstructing the venous circulation. Cirrhosis of the liver is a common cause of ascites. Local dropsies, as a rule, have local causes. *Treatment.*—The cause must be considered and removed if possible. When the dropsy does not yield to remedies the fluid may be removed by paracentesis from serous cavities, and by superficial punctures, or by fine trochars with drainage-tubes attached, in the case of the limbs.

Dropsy, Ovarian.—The gradual distension of the ovary by albuminous fluid. If one ovary only is affected the tumour appears lateral, but afterwards one tumour may extend over the whole abdomen. If both ovaries are affected the tumour appears on both sides, and afterwards central. General health not affected till the tumour causes pressure on abdominal organs, when difficulty of breathing and œdema of feet and legs occur. Cessation of menses usual, but not constant. *Treatment.*—Elastic belt affords relief. Tapping, ovariotomy.

Ear, Diseases of.—*External ear*: 1. *Eczema* occurs to chil-

dren during teething, and to old people ; requires attention to the gums, to the general health, cleanliness, zinc or alum lotion. 2. *Inflammation of the external auditory meatus* often occurs as small abscess or boil. Constant pain, and the passage becomes so swollen as to close the external opening. *Treatment.*—Leeching, poultices, incision with a small bistoury of the abscess in the passage, if it can be made out. In children especially a neglected inflammation becomes chronic and may last months or years. 3. *Hæmatoma auris*, or effusion of blood between the cartilage and its perichondrium, presenting as a dark-coloured swelling on the anterior surface of the pinna, which may become as large as an egg. Often occurs to the insane. May be absorbed, but often ruptures or suppurates ; in the latter event causing deformity. Protection from injury, but no active treatment.

Affections of the middle ear are : 1. *Inflammation of the tympanum*, which may follow, or be combined with inflammation of the passage. *Symptoms.*—Pain and more or less deafness, first from effusion on the tympanum, afterwards from thickening and structural change. Or the disease may go on to, 2. *Purulent catarrh*, and 3. *Perforation of the tympanum*, distinguished, if large, by the passage of air. *Treatment* of purulent catarrh as for inflammation of the passage, careful astringent injections ; for perforation, artificial membrane. 4. *Obstruction of Eustachian tube*, chiefly met with in children who have large tonsils and breathe through the mouth, which is kept open. Mucous membrane of nares and pharynx usually swollen. *Treatment.*—Astringent applications to pharynx, nasal douche, inflation of tube every few days. 5. *Polypus.* Several kinds, more or less fleshy or gelatinous, and which sometimes grow from the edge of a perforation of the tympanum. *Treatment.*—Removal by polypus forceps, and the after-application of caustic to prevent re-growth.

Affections of the internal ear are : 1. *Acute inflammation*, attended by acute throbbing pain, buzzing in the ears, high fever. Matter forms, the tympanum bursts, and the pus escapes. Internal ear is destroyed and deafness results. May be a result of scarlet fever. *Treatment.*—Leeches, fomentations, purgatives, calomel and opium. 2. *Chronic inflammation* occurs as a sequel of acute. Has originated from a blow, causing rupture of the tympanum. *Symptoms.*—Discharge, deafness, dull aching.

Treatment.—Great cleanliness, or in India maggots may form (*vide* p. 564), counter-irritants, attention to general health.

Epilepsy.—The epileptic fit is often preceded by premonitory symptoms, the principal being variability of temper, coldness of the feet or hands, optical illusions, the epileptic aura. The attack may be of every degree of severity, from the *petit-mal* to *epilepsia gravior*. Symptoms of an ordinary attack are after-warning, as headache, pain in limbs, spasms of face, aura, patient falls unconscious, with a loud cry, convulsive movements of limbs and trunk, contortions of countenance, face pale, skin cold and clammy, twisting of head, eyes fixed, pupils dilated, teeth gnashed, foam at mouth, fit followed by sleep. May occur daily or at intervals of weeks, months, even years. Cause unknown. Nothing has resulted from efforts made to establish that epilepsy depends on any recognisable disease in the nervous centres. Often hereditary. Most prevalent from two to twenty years of age. May be excited by emotional causes ; is often connected with insanity ; sometimes seems to depend on gout ; is allied to hysteria, from which it is distinguished by the total loss of consciousness, distortion of the face, solitary cry preceding, and sleep succeeding the attack. *Treatment.*—If aura occurs, a ligature round the limb. Anæsthetics may prevent an attack. Also an emetic, if the stomach is full ; or a lump of ice between the shoulders ; or atropine and morphia snuff. During fit plenty of fresh air, bare chest and neck, prevent person injuring himself or biting his tongue ; afterwards bromides, nitrate of silver, arsenic, and the treatment of any diseased state, as worms, gout, syphilis.

Erysipelas.—Inflammation of the integument, tending to spread indefinitely. Invasion marked by malaise and chilliness. The inflammation often starts from some wound or abrasion ; otherwise from the junctions of mucous membranes and skin, as the corner of the eye. The inflamed skin is bright red, becoming almost white on pressure, with irregular but well-defined margin. The subcutaneous tissue becomes greatly swollen, so that, if on the face, the features are lost. Vesicles form and dry into scabs. In the severe form, or *phlegmonous*, suppuration occurs in the cellular tissue, and sloughs form. In most cases neighbouring lymphatics swell and sometimes suppurate. *Micrococci* have been found filling the lymphatics at the inflammatory margin. Exact *cause* unknown, but it is infectious and inoculable. General bad hygienic

conditions favour erysipelas, which is excited by epidemic influences not understood. *Treatment.*—Purgatives, tincture of iron in large doses, aconite as soon as the temperature begins to rise in minim doses every half-hour; nourishing diet, stimulants if the pulse flags. Locally warm sedative applications, as belladonna extract and glycerine, salad oil and opium, flour. A line of nitrate of silver in front of the advancing rash. Incisions if a soft, boggy feeling indicates suppuration of the cellular tissue. An erysipelatous *lymphaginitis*, and *phlebitis* have been described.

Eye, and Appendages.—1. *Of the eyelids.* A. *Sty*, a boil commencing in the follicle of an eyelash, common in anæmic girls. If seen early, extract the eyelash which passes through it, and apply nitrate of silver. If seen later, foment and open with a fine lancet as soon as pus forms. B. *Blepharitis tinea tarsi*, or inflammation of the whole of the follicles, followed by formation of a crust of discharge on the margin of the lids. If neglected the follicles are destroyed and deformity results. *Treatment.*—The crust should be removed daily by a weak warm solution of bicarbonate of soda, and yellow oxide of mercury ointment applied. Tonics. C. *Ptosis* may be part of general paralysis, or temporary from fatigue or malarious influences, and must be treated accordingly. 2. *Of the Lachrymal apparatus.* A. *Excessive secretion of tears* may be due to irritation from a foreign body in the conjunctival sac, which should be removed; if none mild astringents. B. Impediments to the escape of tears may depend on displacement of the puncta of the lower lid, consequent on conjunctival swelling, or weakness of the orbicularis muscle allowing the lid to fall. But the most usual cause is *stricture of the nasal duct*, when the sac may be seen and felt as a small lump beneath the tendo oculi. This may lead to suppuration, and an opening in the cheek (*lachrymal fistula*). *Treatment.*—In the first case search for and treat the cause. For *stricture* the parts should be emptied several times daily by pressure, and weak lead lotion applied. Radical cure requires the canaliculus to be slit up, and the patency of the passage restored by probes 3. *Conjunctivæ, inflammation of* Varieties: *simple, purulent, gonorrhœal, infantile.* All cause heat, swelling, diffused redness, stinging pain, and often elevation of the conjunctivæ (*chemosis*). All are more or less contagious, purulent most so. *Treatment.*—Simple conjunctivitis, fomentations and

afterwards astringent lotions ; purulent and gonorrhœal, fomentations, strong nitrate of silver solution, relief of tension by incisions from the corneal margin, injections of milk and water to remove discharge ; infantile, warm fomentations. All require protection from light. 4. *Cornea, ulceration of.* Usually presents in scrofulous children as a result of conjunctivitis. Intolerance of light, watering of the eyes, one or two red vessels stretching from the conjunctiva to the ulcer on the cornea. Often leaves a white speck. *Treatment.*—Darkened room, green shade, nitrate of silver to ulcer, quinine, attention to general health. 5. *Arcus senilis* is a crescentic opacity of the cornea, first noticed in the upper part. Distinguished from peripheral zones of opacity, sometimes forming after inflammation, by the arcus being surrounded by an annulus of transparent tissue at the margin of the cornea. It is a fatty degeneration, and when it occurs under forty is indicative of fatty degeneration of heart or arteries. Incurable. 6. *Iris, Inflammation of* : Varieties : *simple, rheumatic, syphilitic.* Conjunctiva inflamed, injected vessels run from circumference to cornea in straight lines. Iris becomes reddish if naturally dark, greenish if blue. Intolerance of light, stinging pain of eye and forehead. Pupil may be blocked by lymph deposit. In bad cases pus forms in the anterior chamber. *Treatment.*—Prevent adhesions by neutral sulphate of atropine (four grains to the drachm). Mercury, especially in syphilitic cases, to slight affection of the gums, local depletion, darkened room, chloral to subdue pain. Pus usually requires evacuation by paracentesis. 7. *Lens* : cataract. Occurs chiefly in elderly people. Lens assumes a white or bluish-white appearance. Patient sees best in twilight, when the pupil is dilated. Operation. 8. *Glaucoma* denotes any condition produced by morbid increase of tension within the eye, by excess of fluids. The increase, and therefore the imperfection of vision, may be gradual or sudden. Degree of tension may be estimated by pressure with the fingers. In most cases the pupil presents a greenish cloudiness. *Treatment.* Iridectomy.

Gastralgia.—Pain in the stomach occurring in various disorders, and must, therefore, be regarded as a symptom and not as a disease.

Gout depends on an excess of lithic acid, being a manifestation of lithic or uric acid diathesis, or *lithæmia* (p. 219). May

be hereditary or acquired. Varieties: *Articular*, affecting the joints, particularly the great toe; *non-articular*, or *irregular*, other parts. Every attack of gout is attended with deposit of urates in the affected tissues, often terminating in chalk-stone. Uric acid may be formed in excess from foods too rich in nitrogen, or from imperfect elimination of uric acid from liver or kidney derangements. An acute attack of articular gout is often excited by exposure. Irregular gout may develope as gastralgia, intestinal colic, palpitation, asthma, chronic catarrh, bronchitis, epileptiform fits, various skin affections, gouty concretions in various parts, chalk stone. *Treatment.*—Acute gout, colchicum with alkalies, mineral water aperients, Dover's powder, or chloral. Locally, wrap the part in cotton wool surrounded with oiled silk; or if the pain is severe saturate the wool with chloroform, or smear with belladonna extract or oleate of morphia. During intervals, moderate diet; stimulants unadvisable; claret, hock, sauterne more admissible than any other. Most careful hygiene. Irregular gout requires special treatment of the malady it developes in. Salts of lithia and potash generally useful. *Retrocedent*, or *suppressed gout*, is metastasis to some internal organ, especially the stomach, marked by sudden acute pain, perhaps bilious vomiting, and tendency to collapse. Inflammation should be excited in the joint first affected by friction or sinapisms; antispasmodics, as ammonia, ether, camphor, when the stomach or heart is affected.

Gonorrhœa.—Incubation, twelve hours to eight days; average, four days. Itching, redness, white discharge, feverishness, swelling of penis, groins, thighs and testicles ache, severe scalding, chordee, thick yellow-green discharge. May give rise to orchitis, cystitis, prostatitis, bubo, phymosis, gonorrhœal rheumatism, warts, gleets, stricture. At the commencement injection of nitrate of silver (one grain to the ounce) or chrysophanic acid (three grains to the ounce) every four hours; aperients, alkalies, hip-baths, fomentations, low diet, rest. When subsiding, copaiba or cubebs.

Gleet.—A consequence of gonorrhœa, but gleety discharge may pass from prostatitis. Urethra inflamed in patches, giving slight resistance to a bullet sound. If fibrous bands form, sound is checked. Ordinarily no pain unless it is prostatic. Zinc injections, and careful diet.

Gravel.—Varieties: *Lithic acid* and its compounds, *oxalate*

of lime, phosphate of lime, triple phosphate. The most common kind, *lithic* acid, seen in form of cayenne pepper crystals, often passing without symptoms, often associated with fulness and heat after meals, eructations, heartburn, depression, malaise. Locally lumbar aching, frequent micturition, irritable bladder. *Treatment.*—Limitation in quantity and quality of food, exercise, alkaline waters. Saline aperients. *Oxalic acid.*—Minute octohedral crystals, or spheroidal ovoid or dumbbell, generally associated with nervous exhaustion, and chronic diseases in which oxidation is retarded. *Treatment.*—Moderate and not rich diet, promotion of healthy action of the skin, tonics. *Phosphates.—Phosphate of lime* shows as plain granules or spheroids of amorphous character; a rare form is stellar phosphate crystallising in minute rods of sheaf-like bundles, grouped in stars or fans. *Triple phosphate* crystallises as transparent triangular prisms with bevelled edges. May show as a flocculent cloud in the urine, or as an iridescent surface pellicle. Urine faintly acid or alkaline. Deposit of phosphates takes place in many diseases having no pathological resemblance, and, therefore, the condition should not be called a diathesis. It is most commonly associated with dyspepsia, and nervous and general debility. The mineral acids are the best remedies, as they diminish the alkalinity of the urine. In long-standing cases different kinds of gravel are often mixed, or alternate. But, as a general rule, red or pink deposits indicate some inflammatory affection, or alternate with gout, and there are seldom symptoms referable to the urinary organs. Yellow gravel is usually attended with pain in the loins, groins, thighs, testicles, with frequent micturition. Sudden acute pain in loin and back with violent sickness, ending after a variable time suddenly with stabbing pain, denotes the passage of a small renal calculus through the ureter into the bladder (*vide* p. 603).

Hay Fever.—A catarrhal affection of the nasal mucous membrane, frequently extending to the pharynx, larynx, and bronchi, often attended with dyspnœa (hay asthma), or with spasmodic sneezing. Induced by vegetable pollen in the atmosphere. *Treatment.*—Removal from the locality, tonic and sedative expectorants.

Hæmatemesis depends on a variety of morbid conditions, the most frequent being ulcer of the stomach. The splenic artery is often the source of the bleeding, which usually takes place after a

meal, and if copious is attended with epigastric weight and faintness. *Congestion of portal system* is also a frequent cause, depending on cirrhosis and other hepatic maladies. Hæmatemesis also occurs in purpura, yellow fever, typhus, jaundice, scurvy. In cases of slight bleeding the microscope may be required to ascertain if the dark colour depends on bile or blood. If in large quantity the blood is dark, mixed with particles of food, and in masses. When hæmatemesis is severe the patient should be maintained recumbent, food must be forbidden, and ice given to suck. If faint, ammonia to nostrils, and brandy as an enema, but not by mouth. The best styptics are gallic acid, alum, acetate of lead; oil of turpentine is often beneficial. When bleeding is slight from probably congestion, a dose of calomel, followed by aperients, with sulphuric acid mixture.

Hæmaturia occurs in many morbid conditions both of the system and urinary tract. If blood comes from the urethra it precedes the stream of urine; if from bladder it comes afterwards. If from the kidneys it is intimately mixed with the urine. *Test.*—When tincture of guiacum and oil of turpentine are mixed equally, and urine slowly added, blood produces an intense blue colour. Hæmaturia occurs chiefly in prostatic disease, Bright's disease, purpura, fever, or as a consequence of parasites. *Treatment.*—Rest, aperients, ice-bags over presumed source of hæmorrhage, astringents as for hæmatemesis, subcutaneous injection of ergotine.

Hæmophilia often occurs during first year of life. Superficial capillary bleeding occurs from different parts, often attended with obstinate swelling of the joints. Those subject to this diathesis suffer after slight bruises or operations, such as division of the frænum, vaccination, or extraction of a tooth. Hæmophilia may occur internally. *Cause.*—Heredity. Styptics are of little use. Tincture of perchloride of iron best internal remedy. Transfusion may be necessary.

Hæmoptysis.—Term restricted to bleeding having its source in pulmonary or bronchial hæmorrhage. Most commonly associated with phthisis or pneumonia. In the former a mere streak, or pints of blood may be expectorated. Blood brought up usually bright, but in cases of very copious hæmorrhage from a large pulmonary branch the blood is venous-looking. In pneumonia expectoration of rusty frothy sputa is characteristic. In doubtful

cases the throat and mouth should be examined, as blood may proceed therefrom. *Treatment.*—Absolute rest, ice, acetate of lead four-grain doses, alum twenty grains, gallic acid twenty to thirty grains, perchloride of iron thirty minims, oil of turpentine, counter-irritation.

Hæmorrhoids are *external* or *internal.* Of the former there are *sanguineous* and *cutaneous.* Sanguineous consist of a soft elevation near the anus, round, livid, and containing coagulum. The cutaneous consist of hypertrophy of the skin, generally left after absorption of the coagulum of the first form. *Internal* piles may consist of one or more dilated vessels. *Causes.*—Sedentary habits, indulgence at table, sexual excess, child-bearing, camel-riding, hepatic disorders. *Symptoms.*—External piles are liable to inflammation and suppuration. Internal piles cause weight, burning in the rectum, pain in the loins, irritable bladder, mucous discharges, leucorrhœa, and many other anomalous symptoms. They are liable to bleed, sometimes excessively. They may ulcerate, or protruding may be constricted by the sphincter and mortify. *Treatment.*—For small piles, moderation in diet, no spirits, exercise, cane seats, avoidance of constipation by mineral waters. For excessive bleeding iced water, injections of tannic acid. When bleeding is frequent and the piles ulcerated, operation.

Heart, Diseases of the. — 1. *Aneurism* is a depression or sacculus formed in the walls communicating with one or more cavities. *Causes.—Inflammation* may lead to ulceration and softening. *Fatty degeneration* may yield to the pressure of blood. *Partial rupture* may take place with hæmorrhage, constituting cardiac apoplexy, with the result of a cyst or sac, which communicates with one of the chambers. *Abscess*, the result of inflammation, may terminate in a sac. *Symptoms* of aneurism are pain, dyspnœa, lividity of surface, palpitation, irregular pulse, various murmurs. But all symptoms may exist with other lesions. *Treatment.*—Palliation of urgent symptoms. 2. *Atrophy. Causes, general*, as marasmus, phthisis, syphilis, cancer, gradual starvation ; *local*, as præcordial adhesions, interference with the circulation of the coronary arteries. *Symptoms.*—Diminished præcordial dulness, feeble impulse, apex-beat above usual position, diminished area of sound, small pulse, feeble circulation. *Treatment* depends on the primary disease. 3. *Dilatation.* When aneurism occurs some part of the heart is dilated, but here is

implied *uniform* dilatation of one or more cavities. *Causes.*—*Obstructive disease at the aortic orifice*, with or without regurgitation. *Continued violent action of the heart* from muscular or nervous influence. *Valvular diseases*, pulmonic or tricuspid, the first causing dilatation of the right ventricle. With dilatation there is usually hypertrophy. *Symptoms* necessarily much the same as from hypertrophy, dulness increased, impulse weak and diffused, sounds feeble, especially the first, which is shortened; mitral or tricuspid murmur implying regurgitation. *Treatment.*—Rest, good hygiene, digitalis in small doses, stimulants if necessary. 4. *Hypertrophy.* The change is an increase in the proper muscular tissue, sometimes also of connective tissue. It may be associated with dilatation. The whole heart, or different parts, may be hypertrophied. The area of dulness is extended, especially the apex-beat; there is slow heaving systolic action, and augmented force of impulse. *Treatment.*—Rest, no alcohol, nitrogenous food, attention to digestion, especial avoidance of flatulence, which embarrasses the heart's action; diuretics if there is tendency to dropsy; sedatives or digitalis, conium, belladonna; if dilatation is present as well, iron and digitalis. 5. *Fatty* (*vide* p. 36). 6. *Fibroid.* A portion of the walls of the heart becomes interspersed with fibroid tissue, sometimes in patches, sometimes forming polypoid tumours. Localised fibrosis may give rise to aneurism. Accurate diagnosis impossible, therefore treatment vague. 7. *Functional disorders.* Distinctive features of functional disorders are rhythmical error, and palpitation without alteration of sounds or morbid murmurs, but they may be associated with increased or diminished impulse. Generally connected with digestive errors, and treatment should be directed thereto. 8. *Inflammation. Endocarditis* generally occurs in association with acute rheumatism. Valves are chiefly affected, resulting in fibrinous deposit. General symptoms are inseparable from the disease with which it is associated. But there may be cardiac pain and shortness of breath. When deposits form a bruit will probably be heard. It is often attended by a friction sound indicating pericarditis as well. *Treatment.*—The medicinal treatment of the original disease, generally acute rheumatism, must be persevered in; digitalis may also be used, and cataplasms over the heart. 9. *Pyæmic Abscess. Symptoms.*—Heart-affection during pyæmia, or after a definite injury, would lead to this

diagnosis. There is no available treatment. 10. *Rupture* is nearly always the result of physical strain. *Symptoms.*—Pain, vomiting, cyanosis, loss of consciousness, and convulsions ; always fatal. 11. *Syphilitic disease* consists of gummata. Heart symptoms occurring in a syphilitic subject would, in the absence of other causes, such as a history of endocarditis or Bright's disease, furnish reason for diagnosing syphilitic disease. Anti-syphilitic remedies in large doses. 12. *Thrombosis.* Coagulation of blood in the heart during life. Coagula found after death are not true thrombi ; they occupy the cavities, and appear as black or red-black clots. Thrombi are situated in the saccular appendages of the auricles, at the apex of the ventricles, and in the recesses between the *columnæ carneæ* ; in other words, as far as possible from the track of the blood-currents. *Symptoms* are præcordial distress and restlessness, cold extremities, stupor or delirium. The impaction of a thrombi in one of the ostia of the heart has caused sudden death. Thombosis cannot be diagnosed with certainty, and the treatment is therefore vague. 13. *Valvular Disease.* Aortic obstruction results from thickening of, or vegetative growths on, the valves. *Symptoms.*—Prominence of præcordial region, forcible impulse to left of normal position. A systolic thrill, loud, rough, rasping murmur, with first sound often extending into the second sound, which is not distinct, heard best at mid-sternum. Pulse regular and slow. Sphygmograph shows line of ascent broken, summit blunt, line of descent has no secondary waves. Epileptiform and syncopal attacks may occur. Hypertrophy of left ventricle remedies this condition for a time ; if this fails the mitral valve becomes affected. If *insufficiency* or *regurgitation* occur, there is a more diffused impulse, the vessels of the neck pulsate visibly, the area of dulness is increased, and a murmur is heard replacing and following the second sound, of a blowing or hissing character. When insufficiency and stenosis are associated there is the 'up and down murmur,' both morbid sounds being present. *Mitral obstruction* results from thickening and rigidity of the valves. The morbid murmur precedes the systole, generally soft and puffing, heard best a little above the left apex and after exertion. Mitral insufficiency affords a loud blowing murmur obscuring the first sound, loudest at the apex, and propagated towards the left axilla. The action of the heart is irregular, and so is the pulse ;

several quick beats following several slow, especially in mitral stenosis. Valvular affections of the heart lead to thoracic and visceral disease, and to dropsy, and are, as a general rule, incurable, although much may be done by mitigating symptoms and careful manner of life. In all diseases of the heart the following rules are applicable: 1. The work of the heart should be lessened by rest, posture, &c. 2. Regularity of the heart's action should be obtained by avoiding excitement, exertion, heavy meals, &c.

Hiccup.—Spasmodic action of the diaphragm, with spasmodic closure of the glottis, excited by irritation of the phrenic nerve. Hiccup may last only a few minutes or for hours or days; is occasionally so intense as to exhaust the person. *Causes.*—Usually indigestion, but present during various diseases of the liver and stomach. Stimulants, bicarbonate of soda if there is acidity, ice swallowed, holding the breath, pressure on the epigastrium, or near internal extremities of clavicles on the throat. When very severe, chloroform-inhalation.

Hooping Cough.—After catarrh, cough of paroxysmal character, consisting of expiratory efforts, cough, and no intervening inspiration until suffocation seems imminent, when a long inspiration takes place attended by the characteristic whoop, caused by the air forcibly entering the still contracted *rima glottidis.* During the intervals catarrhal wheezing. *Cause.*—Regarded as some irritation of the pneumogastric nerve, perhaps from swelling of the cervical glands, in which a special poison has been theorised to reside. Others have considered it a purely nervous affection. Bromide of potash, chloral, belladonna, hyoscyamus, conium, and tonics; change of air.

Hypochondriasis.—The correlative in the male to hysteria in the female. Usually associated with functional liver-disorder, indigestion, or mental worry. Evidenced by morbid sensitiveness, exaggeration of ailments, neuralgic pains, undefined dread and depression, nostalgia. Requires treatment for dyspeptic derangements, fresh air, exercise, change.

Hysteria manifests itself by: 1. *A long train of nervous symptoms*, as flushings, flatulence, palpitations, choking sensations, pains in various parts, clavus, stiffness of joints, paralysis, retention of urine. Nearly every ailment may be simulated, but the pains are always described as terrible. The skin is touched and the patient screams, but firm pressure does not increase pain.

Peculiar drooping eyelids. 2. *Attacks of convulsive fits* are generally preceded by choking sensation, and followed by light copious urine. No insensibility, countenance natural, alternate laughing and crying, patient avoids injuring herself when convulsed. But prolonged paroxysms of semi-hysterical character may depend on albuminuria. *Treatment.*—During fits, fresh air, smelling salts, sal volatile, cold affusion ; afterwards good air and food, attention to bowels, and to menstrual disorder.

Influenza.—Term often applied to simple catarrh, but should be limited to epidemic catarrhal fever, with predominant bronchial or pulmonary disturbance. The first *symptoms* are those of catarrh, with great depression, often followed by bronchitis or even pneumonia. May be complicated with much gastric disturbance. Usually spreads in epidemics from N.W. to S.E. *Treatment.*—That of catarrh, bronchitis, or pneumonia ; liberal diet and tonics.

Intestines, Diseases of.—1. *Atony*, p.142; 2. *Diarrhœa*, p.162; 3. *Dysentery*, p. 189 ; 4. *Inflammation* (*enteritis*) may be caused by irritating substances swallowed, obstruction, cold, some specific poison as diphtheria. *Symptoms.*—Usually diarrhœa with mucus, abdominal pain and tenderness, not so acute as in peritonitis ; temperature may reach 104° ; tongue thickly coated with red papillæ, afterwards red and glazed. Fever remittent in character. Liver usually inactive. In children the disease is ordinarily associated with gastric irritation, evidenced by sickness and sour breath, and it quickly leads to collapse. The child lies with cold extremities, hot abdomen, pinched face, shrunken body, maintaining a short feeble cry. Enteritis, or, as some call it, *muco-enteritis*, *gastric remittent*, or *infantile remittent*, is one of the most important forms of infant mortality. It is scarcely possible to diagnose the exact part of the bowels affected. A *simple intestinal catarrh*, as minor degrees are termed, tends to cure in a few days, but in persons in bad health it may become chronic. *Treatment.*—Castor oil, to remove irritating matter ; milk diet, if digested ; if passed curded, Liebig's raw-meat soup ; lime water. Dover's powder, chloral, antacids, and, for children, early stimulants. Hot fomentations always serviceable. 5. *Obstruction* may occur from intestinal strangulation, caused by false ligaments or bands resulting from previous inflammation ; by twisting, or knotting, or intussusception of the bowels ; by peritoneal pouches ; by openings in the

mesentery; by constriction caused by cicatricial contraction, or compression of the intestines resulting from disease or injury; by peritoneal thickening and contraction; by new growths; by impaction of gall-stones, foreign bodies, or fæcal matter. *Symptoms.*—Constipation, pain, vomiting, which soon becomes stercoraceous, hiccup, abdominal distension, straining, anxiety and distress; probably a tumour in some part of the abdomen; collapse. *Treatment.*—When the existence of obstruction is established, purgatives are inadmissible; opiates by mouth and rectum, enemas, insufflation by means of bellows, hot fomentations, taxis or massage, surgical operations, which are of two kinds, viz., those having for the object removal of cause of obstruction (abdominal section), and those aiming at affording relief by opening above the seat of obstruction (collotomy). 6. *Protrusion* occurs chiefly to children. Bowel protrudes from the anus in the shape of a bright red tumour, as a mere ring, or to the extent of several inches. *Causes.*—Prolonged diarrhœa, frequent purgatives, stone, worms, feeble health. *Treatment.*—Bowel should be returned by pressure with an oiled cloth; child not sit on the stool too long; a band and cork should be worn to prevent protrusion; attention to the causes named. 7. *Stricture.*—If no hernia, it may be cancerous or non-cancerous; the latter comprising contraction following ulceration, inflammation, non-cancerous deposits, injury, effusions of lymph, abscess. *Symptoms.*—At first vague; eventually those of obstruction, when treatment is the same.

Kidneys, Diseases of.—1. *Hyperæmia*, or *congestion*, may be *active*, from influx of arterial blood from inflammation (nephritis), blood poisons, medicinal substances, cold; or *passive*, from hindrance to efflux of venous blood from cardiac disease, imperfections or obstructions in the renal veins, or inferior *vena cava.* Both active and passive congestions are marked by albumen, and tube-casts in the urine. *Active congestion* may pass away, or be succeeded by inflammation of the pelvis of the kidney (*pyelitis*); or of the substance of the kidney (*nephritis*); or by Bright's disease. *Symptoms* of acute congestion are aching of the loins, most on the side affected, and along the course of the ureter, increased by straightening the leg of affected side, and the testicle may be retracted. If inflammation occurs symptoms are more pronounced. If the pelvis is affected the mucous membrane becomes thickened and softened, discharges the angular-tailed epithelial cells with

which it is lined, and eventually pus and blood. *Pyelitis* is distinguished from *cystitis* by absence of vesical pain and frequent micturition, by pain along the ureter, by the more intimate mixture of foreign material with the urine, by the angular cells before mentioned. It is distinguished from renal inflammation by these cells, and by the absence of tube-casts. But as a general rule, when there is *pyelitis* there is also *nephritis*. Pyelitis may be excited in addition to the causes mentioned as productive of hyperæmia, by irritation of a renal calculus, or embolus, or from inflammation of the bladder creeping along the ureter to the pelvis, infundibula, and calyces ; and nephritis may be also caused by the impaction of emboli in the branches of the renal arteries, or from calculi. All the conditions mentioned may result in the secretion of pus, or in the formation of *abscess*, which may be announced by rigors, and is often attended by great constitutional disturbance, the urine becoming opaque, and depositing pus. *Treatment.*—For active congestion or inflammation, leeching, cupping, fomentations, hot bip-baths ; if the urine is scanty, hydragogue cathartics ; if acid, alkalies ; if the reverse, acids. Dover's powder or chloral. Gallic acid is useful in checking discharge of blood or pus. 2. *Calculus.* Most are formed in the infundibula or uriniferous tubes, and are composed of uric acid and oxalate of lime. Eventually the stone is conveyed into the pelvis and ureter. In the pelvis it causes lumbar pain on the side affected, spreading to the front, groin, and down the ureter to the bladder. Aggravated by motion. When a stone passes into the ureter there is intense pain in the loins, and along the ureter to the bladder and testicle, often nausea, vomiting, faintness, urine smoky from blood, or pure blood passes. This may last hours or days, ceasing suddenly when the calculus enters the bladder. *Treatment.*—Hot baths, anodyne fomentations, narcotics in large doses, chloroform. *Preventive.*—Limitation of quantity of food, Carlsbad and Friedrichshall waters for plethoric, Vichy and Ems for less plethoric persons. 3. *Albuminoid disease.* See Bright's disease, p. 584. 4. *Fatty.* Described as a chronic affection, but there is no albuminuria, and it cannot be either diagnosed or treated satisfactorily as a disease *per se.* 5. *Parasites* : A. *Hydatid*, or *echinococcus hominis*, developed from the ova of *tænia echinococcus* ; advance insidious. *Symptoms and diagnosis.*—The discharge of cysts or hooklets, and the development of tumour. B. *Strongylus gigas*, a large nema-

tode worm, from which no distinctive symptoms. C. *Bilharzia hæmatobia*, a trematode worm, very common in Egypt, exciting hæmaturia, and best treated by oil of turpentine. 6. *Syphilitic disease.* Intestinal thickening, or gummatous deposits ; the latter less frequent. Diagnosis of syphilitic disease depends upon the co-existence of renal disease with evidence of syphilis, while other diseases of the kidney are excluded. Bichloride of mercury, and iodide of potassium are the most useful remedies.

Larynx, Diseases of.—1. *Aphonia*, more common in women. Commonly originates from catarrh ; may be purely nervous. Sometimes the consequence of disease of the larynx, ascertained by the laryngoscope. If no disease, attention to the general condition, and to hysterical symptoms. 2. *Inflammation* (*laryngitis*). Cold most frequent cause ; gout and syphilis predispose. The blood-vessels are dilated, and mucous membrane and cellular tissue involved. Often commences as catarrh, followed by feeling of constriction, long-drawn hissing inspiration, husky croaking voice, cough of the clanging croupy character. Expectoration clean and tenacious, *hawked* up ; swallowing painful. Throat shows a little redness. Rise of temperature and pulse. As disease advances suffocative spasms occur, with drowsiness and cold perspirations. The two dangers are spasm and œdema of the glottis, both killing by suffocative dyspnœa. Laryngitis may be confounded with croup or diphtheria, the distinctive sign being the absence of false membrane. *Treatment.*—Equable warm temperature, air rendered moist by vapour of boiling water, poultices or hot sponges to throat, steaming with vapour of iodine and hot water, diaphoretics, leeches to upper part of chest. Should dyspnœa become urgent, tracheotomy. *Chronic laryngitis* may be due to syphilis, or tubercle. *Symptoms.*—As acute in minor degree. *Treatment.*—Of the constitutional malady. 3. *Polypus* may grow from the walls of the larynx. There are seven or eight varieties. *Papilloma* or warty growth most common, usually at the anterior commissure of the vocal cords. *Symptoms.*—Modification of the voice and dyspnœa, but the laryngoscope required for accurate diagnosis. Small papilliform growths may perhaps be removed by caustic ; others require operations. 4. *Spasm* has been referred to as consequent on laryngitis, but occurs independently, as spasmodic laryngitis, spasmodic or spurious croup. Usually occurs to a child with slight catarrh or hoarseness. If no catarrh

noticed, it comes on during the night. The child, after perhaps coughing hoarsely several times, wakes with clanging spasmodic but not husky cough, accompanied by dyspnœa, characterised by the crowing sound with inspiration indicating spasmodic closure of the larynx. Between this condition and that described as the first stage of croup there is every degree. *Treatment.*—As for the first stage of croup (p. 589) ; spasm may also occur as a purely nervous affection. *Laryngismus stridulus*, or child-crowing. With or without previous tendency to convulsions, such as drawing in of the thumbs and great toes, clenching of the hands, squinting ; a child is suddenly attacked with difficulty of breathing, inspiration being accompanied by the crowing sound. When suffocation seems imminent the spasm relaxes, and breathing is re-established. May be due to fright, as from a child being roughly tossed, or to reflex irritation from numerous causes, such as teething, worms, &c. *Treatment.*—Cold affusion on the face and chest, friction, ammonia to nostrils, an emetic, warm bath, artificial respiration. Attention to diet and digestion, bromide of potassium ; if the attacks come on in the night chloral or chlorodyne at bed-time.

Leucorrhœa is a symptom rather than a disease. May be vulvar, cervical, and, as some assert, tubal, from the Fallopian tubes. It is a non-hæmorrhagic discharge of pale colour. Astringents are most useful in checking vaginal leucorrhœa, but the treatment must depend upon the cause and seat.

Lightning, Effects of. — The person struck may be killed outright, and *post mortem* reveal no lesion. Other cases are not distinguishable from concussion. The lightning may cause wounds, broken bones, or patches of erythema, or a curious arborescent figuring on the body. Recovery may occur, or there may result paralysis, or impairment of any of the senses. It has been asserted *rigor mortis* does not occur, which is incorrect. *Treatment.*—Means to maintain the circulation and respiration, cold douche, warmth to the extremities, friction, stimulants.

Liver, Diseases of.—In addition to hepatic diseases treated of as among the diseases of India, the following require notice :— 1. *Atrophy, Simple.* Diminution in size without alteration in structure, except a diminished size of the lobules. *Causes.*—Old age ; inanition either from an insufficient supply of food or from diseases which interfere with the assimilation of food, or from external pressure by tight lacing, pleuritic or pericardial effusions.

There is an absence of any sign of hepatic disease, secreting power remaining, although in a lessened degree. No special treatment. 2. *Acute yellow atrophy; malignant typhoid hæmorrhagic jaundice; contagious jaundice.* After premonitory symptoms of dyspepsia, or rheumatic pains, or without such symptoms jaundice occurs, sometimes confined to the upper part of the body, with a rapid diminution of the size of the liver, and therefore of the area of hepatic dulness. Epigastric pain, vomiting of black material (blood and bile), constipated bowels with cerebral symptoms of the typhoid state, sometimes appearing almost simultaneously with the jaundice, at others two or three weeks afterwards. Hæmorrhages from mucous membranes, and petechiæ common. But although typhoid symptoms are present the temperature rarely exceeds 101°. Urea and uric acid in the urine are much diminished, and *tyrosin* and *leucin* are found. *Predisposing causes.*—Female sex, and pregnancy, in which state the disease causes abortion; dissipation, including drink and venereal excesses; constitutional syphilis; exciting causes regarded as nervous influences, particularly anxiety and grief; malaria; blood poison of typhus and allied diseases; ptomaines; lastly, phosphorus produces symptoms similar to acute atrophy. The occurrence of acute atrophy in an epidemic form has led to the idea that it may depend on some local cause or specific poison; it has also been regarded as contagious. The alteration in the liver is due to a destructive process commencing at the circumference of the lobules, and advancing to the centre, as the result of which the secreting cells disappear, their place being taken by granular matter and oil; hence it is presumed to be an *acute fatty degeneration. Treatment*, unsatisfactory; saline purgatives, diaphoretics, diuretics, warm baths most serviceable. 3. *Chronic atrophy* usually arises from *cirrhosis*, the first stage of which is often hyperæmia and enlargement, after which the organ becomes reduced in size in consequence of destruction of secreting tissue and of the minute branches of the portal vein, by increase of fibrous tissue. After probably an attack of congestion there are dyspeptic symptoms, patient becomes thin and sallow, venous stigmata develope on the cheeks, and there may be dull hypochondriac or shoulder pain. The liver is hard and nodulated (hob-nail), and area of dulness diminished. Next symptoms due to obstructed portal circulation present, viz. ascites, splenic en-

gorgement, enlargement of superficial abdominal veins, hæmorrhoids, hæmorrhages from stomach and bowels, diarrhœa. In almost all cases there is a previous history of spirit-drinking. *Chronic atrophy* may also occur from frequent attacks of inflammation of the capsule, which becomes thickened, and from which fibrous bands pass into the interior. A *red atrophy* has also been described, due to the presence of a large quantity of blood. *Treatment* consists in putting a stop to consumption of alcohol, regular exercise, mineral aperient waters, mineral acids and bitter tonics, the nitro-muriatic bath, nourishing diet. 4. *Cancer*. Premonitory symptoms of deranged digestion as may attend other hepatic maladies, and no diagnosis can be made until tumour occurs, although, as hepatic cancer is often secondary to cancer in some other organ, suspicion may thereby be aroused. When cancerous enlargement projects it is usually irregular, from the presence of nodular formations, but occasionally only one excrescence at a particular part. Swelling hard, resisting, but painful and tender, with paroxysms of lancinating pain. Jaundice often present ; some degree of ascites ; extreme anæmia with chlorotic colour, unless there is jaundice. Rare before 40 ; often a family history of cancer. Distinguished from waxy degeneration by more rapid and painful progress, the absence of enlargement of the spleen, of albuminuria, or constitutional syphilis. From first stage of cirrhosis by the absence of history of alcoholic dyspepsia, morning sickness and venous stigmata on the cheeks. From syphilitic enlargement by previous history, other evidences of venereal, and age. From hydatid by the slow progress of the latter, fluctuation, and hydatid thrill. *Treatment* entirely palliative, with good diet. 5. The *gall-bladder* is subject to diseased conditions, especially to : A. *Enlargement from accumulation of bile.* It then presents as an elastic, pear-shaped, tender swelling in the situation of the gall-bladder. There is generally intense jaundice, absence of bile from the motions, and often enlargement and tenderness of the liver. The tumour may suddenly subside, with passage of much bile. B. *Enlargement from suppuration.* Very tender tumour, no jaundice, motions contain bile, no general enlargement or tenderness of the liver, often history of biliary colic. C. *Enlargement from dropsy* has been described (*hydrops cystidis fellecæ*), which is simply the substitution of a flaky watery material for pus.

D. *Enlargement from accumulation of gall-stones.* Tumour hard and nodulated, painless on pressure, occasionally crackling sensation experienced, or the patient complains of weight; in most cases jaundice, or history of biliary colic. E. *Enlargement from cancerous deposit.* Tumour tender, growth rapid, jaundice and vomiting, cancerous cachexia or cancer elsewhere. *Treatment.*—Enlargement from bile must be treated as jaundice (p. 382); from suppuration, as hepatic abscess; from gall-stones, by constitutional measures directed towards the prevention or dissolving of gall-stones (see below), and cautioning the patient against sudden and severe muscular exertion; if the gall-bladder is tender, opium; lastly, cholecystotomy or removal by surgical operation; from cancerous deposit, by relieving distressing symptoms. 6. *Gall-stones* are composed of cholesterin, and the formation is usually associated with lithic acid, urinary deposits, and gout. Most common in females of middle age, in persons of sedentary habits who consume much saccharine food, and in certain families. Gall-stones may be retained in the gall-bladder, or be impacted in the gall-ducts or within the liver; they may excite inflammation, ulceration, and perforation either in the intestines or in the bile-apparatus, causing abscess, or biliary fistula, or hæmatemesis, or melæna; they may be vomited or passed *per anum*. The most usual result is biliary colic, which often comes on after a full meal or exertion. The pain starts from the epigastrium, shooting to the back, with intervals of comparative ease. During the paroxysm the patient bends forward to obtain relief. There are usually recurring rigors, nearly always vomiting, and, if the attack is prolonged, jaundice. *Treatment.*—If after a full meal an emetic, afterwards hot bath, and opium every three hours in grain doses till the pain subsides; or subcutaneous injection of morphia, belladonna, chloroform, large draughts of hot water, with bicarbonate of soda, two drachms to the pint. Afterwards salts of potash and soda, sulphate of magnesia, chloride of ammonium, salicylate of soda, mineral waters of Carlsbad, Homburg, Vichy, all of which have a questionable reputation for *dissolving* biliary calculi, and a more decided reputation as preventives. Very plain living, no spirits, and a fair amount of exercise. 7. *Hepatalgia*, or *neuralgia.* The occurrence of hepatic neuralgia has been questioned, the symptoms being attributed to pleuritic adhesions, rheumatic pleurodynia, or to stone in the gall-

bladder. But there is no doubt hepatic neuralgia does occur, although not characterised by the acute pain of neuralgia in other parts. *Symptoms.*—Uneasiness, dull pain, or sense of weight, also of the shoulder, which feels tired. Sometimes sharp twitches. These symptoms return at intervals during years, often after exposure. Examination does not detect anything unnatural. The mind dwells on it, and the individual, who is generally hypochondriacal, dreads serious disease. But appetite, digestion, sleep, and bodily condition remain good, and there is no rise of temperature. Medicines of little use; occupation and moderate exercise the best remedies. Iodine paint may be applied to the side. 8. *Hypertrophy simple* is understood to be an enlargement of the liver due to increased size or number of the lobules and of the secreting cells, without alteration of structure. It has been observed in leukæmia and diabetes mellitus. It is, however, rare, enlargements of the liver being nearly always either amyloid, fatty, hyperæmic, hydatid, abscess, or cancer. There are no prominent symptoms of simple hypertrophy. 9. *Hydatid tumour.* There is usually an absence of all constitutional symptoms until enlargement presents, which generally follows one direction. The tumour is elastic, if near the surface there will be fluctuation or 'hydatid vibration.' The surface is smooth, and the growth slow and imperceptible. Diagnosis from abscess (p. 425); from cancer (p. 426). Hydatid is developed from the ova of the *tænia echinococcus*, taken with food or drink. Hydatid tumour may inflame and suppurate, practically becoming abscess of the liver, or it may terminate in the same manner as abscess (p. 426), or it may produce exhaustion and death by pressure on important organs and interruption of their functions. *Treatment.*—Medicines are useless. When the tumour becomes large, evacuation of contents by a fine trocar and cannula, and closure of the opening; repeated if necessary.

Locomotor Ataxy.—First symptoms, unsteadiness of gait, person walks as if intoxicated; heaviness of legs; fatigue after walking; with legs together and eyes shut, would fall if not supported; later on finds he cannot walk without looking at his feet; when upper extremities are affected cannot button his clothes; spasmodic jerking of the muscles; walks with a prancing gait, and brings his heels down forcibly. There may be affections of vision, shifting pains in the limbs, some gnawing, others acute and

lancinating, anæsthesia, paralysis of first, fifth, seventh, eighth, ninth nerves, swelling of joints ; incontinence of urine, abolition of the patellar-tendon reflex, known by no jerking upwards of the foot as occurs in health when a knee, resting on the other, is struck just below the cap. It is a disease of the cord excited by cold, wet, bad diet, onanism, sexual excess, syphilis, suppression of habitual discharges. The spinal membranes are congested, adherent to each other and to the posterior columns, and there is atrophy of nerve-fibre with hypertrophy of connective tissue. *Treatment.*—Equable temperature, generous diet, nitrate of silver in one-eighth grain doses gradually increased to one grain, dry cupping, galvanism, subcutaneous injection of morphia.

Lungs, Diseases of.—1. *Abscess* results from acute inflammation, or pyæmia. No marked physical signs, and no special treatment. 2. *Emphysema* may be *vesicular* or *interlobular*. The first may be lobular, partial lobular, or lobar. All are associated with diseases attended with violent cough. Affected lung feels doughy, pits on pressure, affords little crepitation, altered in colour. Due either to dilated condition of the air cells, or to air in the interlobular tissue from rupture of the sacs. *Symptoms.*—Constant increasing dyspnœa, cough, expectoration, oppression of chest, asthmatic seizures, dropsy. Chest and clavicles become prominent, neck looks shortened, gait stooping, intercostal spaces depressed, ribs prominent, breathing thoracic, tympanitic resonance over whole chest, most marked along anterior borders of lungs, inspiratory murmur faint, expiratory prolonged, râle if there is combined bronchitis. *Treatment.*—Tonics, especially iron, regulation of diet, moderate exercise, warm dry climate. 3. *Hyperæmia*, or *congestion*, may be active or passive. Active hyperæmia is the first stage of *inflammation* or *pneumonia*. But the latter often commences as bronchitis, or with rigors succeeded by pain in the side, dyspnœa, cough, sudden pyrexia. Side pain increased by inspiration indicates implication of the pleura. Respiration ranges from 30 to 60 ; in children may reach 70 ; while the pulse may be only 90 to 120. At first cough is dry, afterwards attended with thin, frothy, tenacious expectoration, which soon becomes rusty or streaked with blood. Delirium may occur. Fine crepitation, heard best at the lower lobes. If not checked, engorgement is succeeded by *hepatisation*, which may be

red, grey, or mixed. Exuded liquids coagulate within the terminal bronchioles, enclosing white and red corpuscles, fibrinous material predominating in red, and white corpuscles in grey hepatisation. Hepatised lung is heavy, sinks in water, cannot be inflated, but is friable. Physical signs—dulness, increased vocal resonance, bronchial breathing. *Symptoms* continue for eight or ten days, when they may terminate by *crisis*, but more usually by *lysis*; the lung is hepatised, gradually assuming its natural condition. Or *chronic pneumonia* may remain; or *abscess* or *gangrene* may result. The *causes* both of hyperæmia and pneumonia are vicissitudes of temperature, especially if combined with overcrowding; but it has been asserted pneumonia is due to a specific cause. Latent (p. 309), asthenic and intermittent pneumonia are described. The diagnostic symptoms of greatest value are cough, rusty expectoration, sudden rise of temperature, without corresponding increase of pulse. In pleurisy no such rapid rise, and pain stabbing. Acute phthisis commences in the upper lobes, and there are marked exacerbations of fever. *Treatment.*—Cardiac failure is the great source of danger, therefore supporting the strength of the patient is the prominent indication, by good nursing, nourishing bland diet, and if necessary, stimulants. The patient should be kept in bed in a well-ventilated room, with equable temperature and free from draught. Vinum ipecacuanhæ may be given occasionally, and morphine at night. Chloral counterindicated, as it depresses circulation. Quinine may also be used with the view of lessening pyrexia. The cold bath has been used, but this is not recommended (p. 317). Linseed poultices may be applied to the chest. If delirium is a marked feature hyoscyamus and bromide of potassium. Constipation in the earlier stages requires a purgative. The mouth may be cleansed of viscid sputa by glycerine and lemon-juice. *Hyperæmia, passive*, of the lungs depends on other diseases, or on exhausted conditions, and is most seen in the lower posterior lobes. 4. *Chronic pneumonia* is usually the result of acute, when the hepatisation is not fully resolved, and it is usually complicated with some degree of bronchial affection (*broncho-pneumonia*). But chronic pneumonia may originate insidiously, when the symptoms are obscure, consolidation occurring without rusty sputa (p. 309). 5. *Tuberculosis.* See Phthisis.

Lupus.—Two forms described, *erythematosus* and *vulgaris*.

They are merely variations ; both depend on the formation of a new cell growth in the cutis, followed by interstitial absorption, epidermic exfoliation, scab, ulceration, and scar. Both attack the face preferentially, and the ætiology is unknown. *Lupus erythematosus* commences with patches of purplish redness on the cheek or nose, which become covered with crusts. Lupus vulgaris commences as a reddish-brown blotch or blotches the size of a pea, which may be raised; from this epidermic scales are thrown off (*Lupus exfoliativus*) ; or destructive ulceration may set in (*Lupus exedens*). Both forms are very slow in progress, lasting for years. *Treatment.*—In the milder form attention to the general health, arsenic, lead lotion, iodine paint, mercurial ointment. In the ulcerative form, caustics, solid nitrate of silver bored freely into all the ulcerated parts. If not successful, acid nitrate of mercury painted on with a glass brush. After dressing, zinc ointment, nourishing diet.

Measles.—Coryza, cough, sneezing, enlargement of glands of neck, fever. On the third day, increase of symptoms as above, with conjunctivæ injected and tonsils enlarged. On the fourth day the rash, red circular spots raised and rough, first forming crescentic groups, which coalesce into patches of irregular outline. Face first covered and may be swollen, afterwards neck, chest, body. There is a peculiar smell. Rash commences to decline within thirty-six hours in the order of invasion, with desquamation of the cuticle. On the dark skin the eruption appears yellowish, and lighter in colour than the skin. The declining rash may leave a mottling not unlike typhus eruption. Measles in scorbutic persons may assume a form known as *black* or *hæmorrhagic*. The accompanying coryza may run on to bronchitis, laryngitis, or broncho-pneumonia, which may also present as sequelæ. Tendency to sudden elevation of temperature with reappearance of rash has been noticed for a fortnight in children, measles thus showing an affinity to dengue. Measles is highly contagious ; personal infection begins before the rash appears, and is not over for at least a month. It lasts much longer in clothing and closed rooms. Peculiar rod-shaped, highly refractive microscopic bodies have been found in the breath and tissues, which may be the poisonous germ. *Treatment.*—Rest, pure air, equable temperature, nourishment, diluents. *Measles, German*, (*Rotheln, rubella, rubeola sine catarrho*) : the spots are brighter

in colour and more discrete, are preceded by one day of headache and fever, or slight sore-throat, and there are rarely severe symptoms.

Menses, Disorders of, are classed as amenorrhœa (deficient), dysmenorrhœa (painful), menorrhagia (profuse menstruation). Amenorrhœa depends on general or local states. *General* conditions are those acting unfavourably on nutrition, such as the demands made on the system by the advent of puberty under poor food and bad hygiene ; or by exhausting diseases, as phthisis and albuminuria. Cold may arrest the discharge. *Local* conditions are imperfect development of the ovaries, imperforate hymen, or closure of the *os uteri* or vaginal orifice from other causes. *Treatment* of the first form is the treatment of the general state. Local conditions generally require surgical interference. *Dysmenorrhœa* may be *mechanical*, from flexions of the uterus ; *congestive* or *inflammatory*, generally the result of mechanical, or originating from abortion or labour ; *neuralgic*, generally associated with malarious saturation of the system ; *membranous*, in which shreds, patches, and sometimes a cast of the uterus is expelled ; *ovarian*, due to the growth and rupture of Graafian follicles ; *gouty*, due to the lithic acid diathesis. *Symptoms.*—Tenderness and pain in the loins, shooting down the thighs, nausea, diarrhœa, frequent micturition. *Ovarian dysmenorrhœa* is further characterised by pain generally in the left ovary coming on before the catamenia, when examination *per vaginam* may detect a tumour or fulness. *Treatment.*—Hip baths, fomentations, antispamodics ; afterwards iron and other tonics, with great attention to general health. *Menorrhagia* occurs both during catamenial periods and at other times. Hæmorrhage sometimes profuse. Bleeding may arise from tumours, polypi, moles, scurvy, the absence of which should be ascertained. *Treatment.*—Recumbent posture, ergot, gallic acid, acetate of lead, if necessary plugging of the vagina.

Mesenteric Glands are liable to congestion or inflammation, usually connected with typhoid and dysentery ; also to scrofulous or tubercular disease, and to *lymphadenoma* (p. 39). The term *tabes mesenterica* is given to the tubercular or strumous enlargement of the mesenteric glands ; also *abdominal phthisis*. Enlargement of mesenteric glands occurs in atrophy, of which it is often the cause (p. 580). *Symptoms.*—Pain, swollen abdomen, veins distended, body thin and wasted, sour diarrhœa, temperature

increased, hectic, exhaustion. Often enlarged glands may be felt. *Treatment* general, according to cause, with medicines to check diarrhœa, tonics, and stimulants.

Miliary, *milk fever*, or *weird*, may attend the secretion of milk which is delayed. Chilliness, followed by feverishness, pain, and soreness of the breasts. Should the breasts become flabby, discharges lessen, or the abdomen become tender, puerperal fever or peritonitis may be feared (p. 622). *Treatment.*—An aperient, fomentations, protection from chill.

Mouth, Ulcers of the, may be due to dyspepsia, to aphthæ, to venereal, to scurvy, to mercurial salivation, and must be treated constitutionally. Best application, dilute sulphuric acid.

Mumps.—Contagious inflammation of the parotid gland, marked by swelling under the ear and chin, with difficulty of swallowing and fever. Testicles and breasts may be affected. Hot fomentations and aperients.

Œsophagus, Diseases of.—1. *Inflammation*, usually caused by irritating or corrosive substances, but may be extension of catarrh, croup, diphtheria. *Symptoms.*—Burning pain and thirst; swallowing causes agony. *Treatment.*—Ice to suck; for nourishment nutritive enemas, morphia subcutaneously, opiate fomentations. 2. *Ulceration* may result from œsophagitis, when the pain is more localised and often referred to between the scapulæ and top of the sternum. Patient's strength to be supported by nutritive enemas, or he may be fed with a small tube. 3. *Stricture* may result from former conditions, or may be cancerous, non-cancerous, spasmodic, or hysterical. *Symptoms.*—Difficulty of swallowing, coming on gradually, except in spasmodic or hysterical, when it comes on suddenly. Food passes to a certain extent, and returns with much mucus. In cancerous stricture there is great pain, enlargement of lymphatic glands, and probably a history of cancer in the family. Most strictures are also attended by cough and husky voice, caused by pressure or irritation of the trachea, or recurrent laryngeal nerves. If doubt exists as to diagnosis, passsage of a bougie. Spasmodic stricture is often associated with hysteria or hypochondriasis. *Treatment.*—Periodical passage of bougies for non-cancerous stricture; feeding with a small tube; for cancerous stricture, nutritive enemas. For spasmodic or hysterical stricture, general treatment for hysteria.

Ovaries, Diseases of. — 1. *Acute inflammation.* Pain over

the pubes, tenderness in one iliac region, irritable bladder. Ovary may be felt swollen on vaginal examination. *Treatment.*—Rest, with hips raised and thighs flexed ; mustard poultices, bromides, Dover's powder, or chloral. 2. *Chronic inflammation.* Symptoms as in the acute form, but less intense. Generally recurs at menstrual periods, as *ovarian dysmenorrhœa* (p. 613); may follow the acute form, or originate from gradual hardening and enlargement of the ovary from clots in some of the ovisacs, or from adhesions between the surface of the ovary and the Fallopian tube or fimbriæ, consequent probably on extension of gonorrhœa, or on abortions. *Treatment.*—Attention to the general health ; sedatives, especially bromides. In cases of falling down of the ovary, an elastic ring pessary. Extirpation of the ovary has been advised. 3. *Ovarian tumour* may be solid, semi-solid, or cystic, containing albuminous fluid (*vide* Dropsy, p. 589).

Pancreas, Diseases of.—1. *Hypersecretion* has been supposed to give rise to a form of pyrosis, the pancreatic juice entering the stomach, or there may be a constant spitting of a fluid like saliva. Diarrhœa alba (p. 168) was once supposed to be caused by overflow of pancreatic secretion (*fluxus pancreaticus*). 2. *Deficiency of pancreatic juice* has been presumed to be characterised by the presence of free fat or oily matter in the stools. 3. The pancreas is also liable to *fatty degeneration* and to *obstruction* of its ducts by cystic formations, also to *cancer*; but the symptoms are vague, and treatment unsatisfactory.

Paralysis may be of *encephalic*, *spinal*, or *peripheric origin*, but instances occur where the paralysis has a *simultaneous*, *encephalic* and *spinal origin*. The first may originate in apoplectic or epileptiform fits, and is usually confined to one half the body. Paralyses of spinal origin commence without fits or impairment of consciousness, and implicate to a variable extent both sides of the body, especially the lower parts. Peripheric paralyses result from injury or disease of nerve-trunks, and are recognised by the loss of power being limited to the muscles supplied by the nerve affected. *Paralysis agitans* is a disease of advanced life, considered due to cerebral *sclerosis*, when trembling occurs before paralysis, and to cerebro-spinal sclerosis, when paralysis is noted before trembling. The subjects of paralysis agitans run or plunge towards an object, while unable to walk slowly (festination). *Writer's cramp* is a semi-paralysis of the flexors of the

hand consequent on much clerical work, and the only means of cure is rest. Paralytic affections are so frequently associated with diphtheria, that they have been regarded as peculiar, and as part of that disease, which is questionable. *Facial paralysis* is due to disease, irritation, or injury of the nucleus or fibres of the *portio dura* of the seventh nerve. Most common causes of facial paralysis: cold (p. 440), rheumatism, syphilis, diphtheria, bone-disease. Fluids run out of the mouth, the eyes cannot be closed even during sleep; at rest, the forehead is smooth, the angle of the mouth lowered; on movement, half the forehead only moves; in smiling, lips are displaced towards healthy side. Whistling impossible. Loss of taste, obliquity of uvula. *Treatment* of all kinds of paralyses depends on the cause.

Pelvic Cellulitis, or inflammation of the cellular tissue surrounding the pelvic organs, most frequent in the female. May originate from any irritation of the uterus, vagina, or rectum; most frequently from an extension of uterine malady, or after abortion or confinement. Exudation of albuminous nature takes place, which may end in absorption or abscess. *Symptoms.*—Tenderness on deep pressure, dull aching pain in defæcation, dysuria, pain down the legs, especially on movement of the thighs, pyrexia; in some cases tumour detected *per vaginam*. But often symptoms little marked, particularly when it occurs insidiously after parturition. If abscess forms, it may be attended by rigors, and will probably make its way *per vaginam*. *Treatment.*—Salines, sedatives, fomentations, warm injections *per vaginam*; afterwards iodide of potash and the vaginal douche.

Pelvic Peritonitis is by some authors distinguished from cellulitis, chiefly from more acute tenderness, a higher temperature, no pain of thighs, no suppuration or tumour, tympanitis. But pelvic peritonitis may terminate as cellulitis.

Pericardium, Diseases of.—1. *Dropsy*. This is a possible occurrence in all diseases where there is tendency to transudation of serum. It may present as a sequel of inflammation. When effusion occurs the pericardial sac assumes a pyramidal form, and there is dulness from the second left costal cartilage to the lower edge of the sixth rib. 2. *Inflammation* occurs most frequently in connection with rheumatism or Bright's disease. At first there is rough vascularity, and afterwards fibrinous effusion, perhaps

stained with blood (hæmorrhagic pericarditis). Occasionally the effusion contains pus. *Symptoms.*—Anxiety, oppression, pain, palpitation, delirium. Delirium in acute rheumatism, and sudden cardiac spasms or coma in renal disease, indicate pericardial affection. At first a 'to and fro' friction sound, resembling the rubbing of paper or creaking of leather, and not like a valvular murmur (p. 599). Friction fremitus sometimes detected. Irregular action of heart. Displacement of apex-beat upwards by the effusion. *Treatment* regulated by the nature of accompanying disease. A blister may be applied, but local applications are not of much use. When the amount of fluid is large and symptoms urgent, paracentesis has been advised.

Peritonitis may occur from numerous causes, but acute peritonitis generally results from cold. Invasion usually indicated by rigors, followed by acute pain, commencing locally, and described as hot, cutting, shooting, boring. Movement, pressure, deep inspiration increase this pain. Patient lies on the back, with legs drawn up, and respiration is thoracic. Nausea and vomiting set in early; usually constipation. Micturition frequent. At a later date tympanites. Marked depression throughout. The products of the inflammatory process are fibrinous exudation, or organisable lymph. *Treatment.*—After clearing the bowels by castor oil, opium in half or one-quarter doses every four hours. There is a tolerance of opium in this disease, and it does not seem to lessen peristaltic action. Effervescing medicines and hydrocyanic acid, or drop doses of creasote if vomiting is urgent. Hot fomentations, nourishing liquid food, and stimulants. 2. *Peritonitis, chronic*, may either result from an acute attack, or set in gradually, when it is usually part of some degenerative process, generally strumous. It is often connected with disease of the mesenteric glands (p. 613), or with phthisis. *Symptoms* are loss of appetite, nausea, emaciation, diarrhœa, hectic fever. Fluid is effused and adhesions form, on account of which the fluid does not gravitate so freely as in ascites, which with thickening of the peritoneum, which may possibly be felt, distinguishes from ascites. A friction sound sometimes heard during respiration. *Treatment.*—Fomentations, counter-irritants, iodide of potassium, iodide of iron, medicines to check diarrhœa, nourishing diet. 3. *Peritoneal dropsy.* See Ascites.

Perityphlitis. — Inflammation of the tissue behind and

around the cæcum, but the latter organ is always implicated. It may be acute or insidious (*vide* p. 145).

Pharynx, Diseases of.—1. *Acute inflammation.* Chilliness, feverishness, sore-throat, dryness of the throat, constant hawking, swollen tonsils and uvula, much secretion of mucus, often deafness from implication of the Eustachian tube. If the inflammation becomes localised in the tonsils it constitutes *quinsy*, when suppuration occurs, causing increased and throbbing pain, headache, difficulty of breathing and swallowing. *Treatment.*—Fomentations, steaming, ice to suck, tincture of aconite in drop doses every quarter hour for two hours, afterwards every hour; if suppuration occurs calmative doses of chloral, lancing the abscess, from which thick fœtid pus escapes, affording immediate relief. 2. *Chronic*, or *follicular inflammation.* The mucous membrane is reddened, rough, puffy, or presents a mammillated appearance from the mucus follicles being enlarged, with much secretion of tenacious mucus, giving rise to hawking, spitting, and pricking in the throat. If neglected the discharge may become mucopurulent. Often depends on secondary syphilis, or stomach disorders. Those speaking much, as clergymen, often suffer from chronic follicular inflammation under the term 'relaxed throat,' but there is nearly always some dyspepsia. *Treatment.*—If no specific cause, tonics, exercise, prohibition of smoking and public speaking. Locally, sprays, swabbing with nitrate of silver solution (five to ten grains), or alum (ten to thirty grains). Gargles do not reach the parts. Change of air and occupation.

Phlegmasia Dolens usually occurs to lying-in women. There is always phlebitis and often thrombosis, and it has been attributed to obstruction of lymphatics. Commences with pain and tenderness in the course of the femoral or external saphenous veins, while sometimes thromboses may be felt. Then elastic hard swelling, not pitting on pressure, appears, frequently commencing above and spreading downwards to the foot. Sometimes there is an erythematous blush along the course of a vein. The colour of the limb is pale, hence called 'white leg,' or 'marble leg.' Power of voluntary motion is almost lost. After nine or ten days it gradually subsides, but there is often a sequela of persistent or intermittent aching, œdema of the ankles, deficiency of muscular power, or hypertrophy of tissue, similar to elephantiasis

(p. 246). One or more of these conditions may become permanent, causing ulcer of the leg. *Treatment.*—Opiates to procure sleep ; limb to be kept at rest, anodyne fomentations. After the acute stage, and when danger of moving thrombi ceases, frictions, with mercurial and belladona ointments, bandages, diligent use of the leg.

Phthisis, or *consumption,* is characterised by cough, mucous expectoration, loss of colour and strength, emaciation, night sweats, loss of hair, quickened pulse, evening rise of temperature, pain in chest, increased respiration. As the disease advances yellow expectoration, occasionally streaked with blood, and eventually pus. Hæmoptysis may or may not occur, but usually does do so ; often diarrhœa. *Causes.*—Hereditary, debilitating conditions, mental depression, bad ventilation, moist cold climates, dampness of soil, all of which tend to the formation and deposit of tubercle, which is an opaque, dirty white, homogeneous substance, commencing from distinct foci, the result of exudation from the blood. In some cases the lungs are filled with miliary tubercle from apex to base ; sometimes there are local aggregations. As these latter soften down cavities or *vomicæ* are formed in the lungs. The physical sounds vary much. Generally dulness and bronchophony, except over a superficial cavity. A cavity is revealed by pectoriloquy, crepitation of coarse character, clicking and croaking sounds, and is accompanied by opaque morning nummular sputa, increased night sweats, and hectic. Hæmorrhage usually proceeds from giving way of an artery in a cavity. Phthisis has been divided into varieties, as *acute tuberculosis, tuberculo-pneumonic, catarrhal, fibroid, hæmorrhagic, laryngeal, chronic, latent* (p. 313). *Treatment.*—Principally dietetic and hygienic, the object being to introduce as much nourishing food as can be digested, and to place the patients in well-drained and ventilated houses on dry soil, flannel being worn, and as much exercise (the best of which is riding) as can be taken without fatigue ; medicines to meet symptoms ; change of climate.

Pleuritis.—*Cause,* cold. Sets in with rigors and fever, with acute stabbing pain about the false ribs, increased by cough and inspiration, so that ribs of affected side are more or less immovable. At first a friction sound may be heard, which ceases when effusion takes place. Several varieties described as *dry,* when there is little or no effusion ; *diaphragmatic,* when neither

friction sound nor effusion present, and the ribs move naturally ; *tubercular*, when connected with phthisis ; *pleuro-pneumonic*, when with inflammation of the lungs ; *fibroid*, when thickening of the pleura compresses the lungs ; *chronic* or *quiet*, when it occurs insidiously ; *latent*, when there are no marked symptoms (p. 311). Result of pleurisy is effusion, which may end in *absorption*, *adhesion*, *hydrothorax*, *empyema*, *pneumothorax*. Absorption is equivalent to cure. Adhesions probably give little or no future trouble, or may cause *pleurodynia*. One or other of these results are usually obtained by leeches, tartar emetic, if the patient is young and robust, Dover's and James's powders, chloral at night, and bandaging the chest. When effusion occurs, hydragogue aperients, iodide, ointment of oleate of mercury. But if effusion rises above the angle of the scapula and remains there, paracentesis may be required. *Symptoms of effusion* are dulness, loss of elasticity of thoracic wall, absence of respiratory sounds, and of vocal thrill, sometimes bulging of intercostal spaces. Known from consolidated lung by alteration of dulness from different positions, and by ægophony. The nature of the fluid can only be ascertained by exploration. *Pneumothorax* usually results as a consequence of empyema. Physical signs are enlargement of side, tympanitic sound, and no movement. In pneumothorax with bronchial fistula the sharp line between resonance and dulness may change after profuse expectoration, and the entrance of air may be detected. There is also amphoric cough resonance ; the metallic ring or echo from vocal or cough vibrations of the fluid ; the metallic tinkle from bursting of bubbles on dropping, and succussion. For pneumothorax, no specific treatment ; if respiration is embarrassed a fine trocar and cannula may be used.

Pleurodynia.—A name given to rheumatism or neuralgia of the chest walls. But similar pain may occur from adhesions remaining after pleurisy, or from gall-stone or other hepatic maladies (p. 608), or from syphilitic periosteal affection of the ribs. *Treatment.*—According to presumed cause.

Pregnancy, Diseases of, are 1. *Indigestion* ; 2. *Fainty feelings and palpitation* ; 3. *Morning sickness* ; 4. *Toothache and salivation* ; 5. *Swelling and cramps of the legs* ; 6. *Varicose veins* ; 7. *Irritation of the breasts* ; 8. *Hæmorrhoids* ; 9. *Irritation of the bladder and private parts* ; 10. *Miscarriage*. All these ailments depending on

the pregnant condition cease when that is over, and must be palliated on general principles, and as if occurring without pregnancy. None are ordinarily dangerous except miscarriage, although the sickness of pregnancy has sometimes been so severe as to justify the production of labour. *Miscarriage* commences with pain in the back, loins, and hips, with bloody discharge. Then pains like those of labour, often vomiting, and sometimes profuse bleeding. *Treatment.*—Quiet, coolness, recumbent posture, cold applications, sulphuric acid and opium mixture, and if profuse hæmorrhage occurs, removal of the ovum.

Progressive Muscular Atrophy usually first appears in the upper right extremity, and in the muscles of the thumb and hand, which looks like a bird's claw. It afterwards involves any or all muscles, even of respiration and deglutition. There is loss of power, movements are awkward, and as in locomotor ataxy there is loss of muscular co-ordination. There are also cramps, tremors and twitches, sometimes anæsthesia, or pain in affected parts. Tongue may be shrunken, and articulation imperfect. When the muscles of deglutition are affected cough is excited by liquids, and the person often dies from a bronchial attack, being unable to expectorate the mucus. It depends on granular, fatty, or waxy degeneration of the muscles, and in atrophy of the nerve cells in the anterior cornua of the spinal cord, resulting from pigmentary and granular degeneration. Exciting causes not known, but often connected with syphilis. *Treatment.*—Warm clothing, warm baths, iodide of potassium, arsenic, and other tonics, cod-liver oil, phosphorus and strychnine, galvanism.

Prostate Gland, Diseases of the.—Most common malady, inflammation, or *prostatitis.* May be caused by gonorrhœa, or by stone, masturbation, excessive coitus, piles. *Symptoms.*—Weight in the perinæum, frequent micturition, heat or burning in the rectum, feverishness. May result in chronic prostatitis with enlargement of the gland, most common in advanced age, and characterised by increasing slowness and difficulty in making water, a sense of weight and straining, so that the patient often imagines he has piles. Next the bladder becomes irritable, hypertrophied, sacculated, with frequent micturition. The enlarged gland mechanically prevents the bladder being perfectly emptied, and the urine remaining decomposes and becomes ammoniacal, setting up chronic cystitis. Then the urine is loaded

with sticky tenacious mucus which adheres to the vessel, and is frequently tinged with blood. There may also be fits of retention. A sound traverses the urethra to the bulbo-membranous part, and then meets with resistance. *Irritable* prostate has also been described, but this is always connected with some amount of congestion. *Treatment* of prostatic congestion or inflammation, hip-baths, fomentations, leeches, alkalies, rest. When the prostate is enlarged, bowels should be kept open so that there may be no straining at stool. If the urine is acid, alkalies, as bicarbonate of potash ; if alkaline, acids ; if ropy and thick, benzoic acid with carbonate of ammonia. Enemas of warm water for occasional spasmodic pain or retention, or suppositories. In advanced cases the use of the catheter, and washing out the bladder.

Puerperal Diseases.—1. *Convulsions*, are of an epileptiform character, occurring either in the latter months of pregnancy, during labour, or after delivery. Often preceded by frontal headache, derangements of vision, puffiness of eyelids, face, or ankles, in which case the condition will be probably associated with albuminuria. Œdema of any part except the feet in a pregnant female should be viewed with suspicion, as the probable forerunner of convulsions. The attack differs little from an epileptic fit, and often excites premature labour. It is attributed to irritation of the vaso-motor centres, from retention in the blood of matters (p. 633) which should be eliminated by the kidneys. *Treatment.*—Patient should be protected from injury, or from biting her tongue. If the woman is of full habit, the face congested, and the pulse bounding, venesection, enemas, chloral, chloroform, subcutaneous injection of morphine, afterwards bromides. 2. *Puerperal fever* comes on three or four days after delivery, and depends on poisoning of the blood from absorption of putrid matter from the womb. Shivering, fever, perspirations, breasts become flabby, discharges lessen or cease, irritating diarrhœa with perhaps scybalæ, abdominal tenderness, swelling of the large joints, fever assumes typhoid type. If it commences early, may at first be mistaken for miliary fever (p. 614), but usually commences after, not with secretion of milk. *Treatment.*—Purgatives, enemas, injections of warm water into the vagina, fomentations to bowels and breasts, diaphoretics, good ventilation, disinfectants. 3. *Puerperal peritonitis* may or may not be associated with puerperal fever. Symptoms and treatment

as for puerperal fever, or peritonitis. 4. *Puerperal insanity* generally takes the melancholic form, and usually seems connected with anæmia. Induced by disorders of nutrition, rapid succession of pregnancies, prolonged lactation, hæmorrhages, especially if such causes are imposed on heredity. Occurs during parturition, or within six or eight weeks. Indications of treatment : to remove all causes of irritation, obtain repose, and restore strength. The milk, breasts and lochiæ must be attended to ; room should be quiet and darkened ; patient under constant supervision. Chloral is the best hypnotic. Generous diet and stimulants.

Pyæmia.—Caused by entrance into the blood of an animal poison, originating in a wound or local inflammation. It consists of two morbid processes ; the first manifested by general constitutional disturbance, the second by secondary lesions. Exact nature of the poison unknown ; probably a ptomaine. Attack usually sudden, with rigors, pain in the limbs, and fever of remittent type ; the skin may become jaundiced, and typhoid symptoms soon develope. In less acute cases pain and swelling of joints, subcutaneous abscesses, or purpuric patches present, and pneumonia may occur. After the lungs the liver is most likely to be affected by secondary deposits. When pyæmia results from a wound the discharges stop, and the wound and surrounding tissues present an unhealthy infiltrated appearance, and neighbouring veins are blocked with coagula. Overcrowding and other bad sanitary conditions predispose. Probably the most useful medicine is quinine in large doses. Secondary abscesses should be opened early, and treated on ordinary surgical principles. Bed-sores must be guarded against by generous diet and every hygienic precaution.

Rheumatic Fever has been attributed to lactic acid in the system ; to entrance of malaria into the blood, the joint symptoms being secondary. Also supposed to originate from gonorrhœa. But the most usual exciting cause is damp cold. *Symptoms.*—Fever, the joints swell, are red, and acutely painful, the condition quickly subsiding in one part and reappearing in another Sour perspirations. Temperature rising to 105° indicates complication, which may be *cardiac* (p. 589), *pleuritic* (p. 620), *pneumonic* (p. 610), *nervous cerebral rheumatism*, or *rheumatic meningitis*. *Treatment.*—Good nursing, warmth to the affected joints by the application of cloths steeped in strong hot solution

of carbonate of soda, salicylate of soda in 25 grain doses. Nearly every kind of medicine, local application, and counter-irritation have been used. *Rheumatism, chronic*, may remain after an acute attack, or may develope gradually. Leading symptoms are pain and stiffness of joints, aggravated by cold, when the symptoms increase and swelling may occur. Chronic rheumatism in a severe degree has been described as a distinct disease, as *rheumatic arthritis*; but the only distinctions are the greater amount of deformity present and the less liability to the involvement of certain joints in the latter; differences not sufficient to separate the malady into distinct diseases when there are so many features of similarity. In both forms, after the disease has been continued some time, there is great deformity of the joints, which become more or less useless. *Treatment* consists of friction with stimulating liniments, warm rollers, iron and cod-liver oil, alkalies; when costiveness occurs mineral waters. *Rheumatism, muscular*, may be sub-acute or chronic. Some muscle or set of muscles is affected. The part is painful only when the muscles are somewhat tender. There may be slight pyrexia, furred tongue, &c. Varieties: 1. *Stiff-neck*, when the sterno-mastoid is chiefly affected; 2. *Pleurodynia*, when a particular intercostal space is affected, the ordinary signs of pleural, pulmonary, and cardiac disease being absent; 3. *Lumbago*, when the muscles of the loins are affected; 4. *Cephalodynia*, when the scalp muscles are painful, especially on movement; 5. *Dorsodynia*, when the scapular muscles are affected. *Treatment.*—Warm baths, alkalies, colchicum, iodide of potassium, counter-irritants, and anodyne applications, warm clothing, dry soil and habitations.

Rickets arises from impaired nutrition in childhood, induced by bad feeding, bad air, damp rooms, want of sunlight, exercise, and cleanliness. Hereditary syphilis is a predisposing cause, scrofula not. *Symptoms.*—Digestive derangements precede, then sweating of head, face, and neck. Child dislikes to be covered, or to be touched, and seems to dread movement. Ends of long bones enlarge, flat bones become thickened, and all lose firmness. Child is unsteady on its legs or unable to walk, is drowsy by day, restless by night, constantly moving the head about. Motions loose, pasty-looking, and offensive. The soft bones now yield and deformity occurs, the direction of the bending depending on the force, either muscular or otherwise, applied.

The skull becomes enlarged, and the face seems small ; the spine, ribs, pelvis are also implicated, and may become deformed. The precise nature of the changes in the bones depends on growth being retained by slow ossification of the cartilaginous structure, while abnormal development of cartilaginous structure and of the fibrous periosteum takes place. *Treatment.*—Hygienic and dietetic, avoiding farinaceous foods.

Scarlet Fever.—An infectious specific fever, sometimes commencing with chilliness, at others suddenly, the lymphatic glands at the angles of the jaw enlarging, and the throat being red. These symptoms increase, and on the second or third day the rash appears as a multitude of minute scarlet points. The skin itches, and the tongue is furred with red papillæ. The tonsils are scarlet swollen, and often coated with white mucus. Rash declines on the fifth day, with desquamation. The varieties are *minor* and *gravior* ; or *simplex* and *anginosa* or *maligna*. The worst form consists of sore throat without rash, which may be mistaken for diphtheria. *Cause* is some product of the sick conveyed to the unaffected, and supposed to be spores, most abundant in the breath of the patient. Period of incubation four or five days. Infection attaches during the full period of the disease, and for six or eight weeks afterwards. Clothes unpacked months after will give infection, and also closed rooms. Milk absorbs the poison of scarlet fever, and conveys the infection. Persons protected by a previous attack may have sore throat in an infected house, and this sore throat is capable of conveying the infection. Sequelæ of scarlet fever are anasarca with albuminous urine, ophthalmia, discharges from the nostrils, diarrhœa. *Treatment.*—Patient should be kept moderately cool in a well ventilated room, and be allowed good broths, &c. Throat should be steamed, powdered alum blown in, and ice given to suck. Excreta should be promptly removed. Medicines are useless. If afterwards the skin peels off, sweet oil or glycerine. Dropsical swellings or other after effects must be treated as mentioned under the headings.

Sciatica commences often with stiffness of the limb, followed be shooting, darting, or burning pain, often paroxysmal. Parts usually affected are buttock, back, or thigh, knee, front, back, and outside of leg, foot except inner border. Sometimes the pain may be traced in the course of the nerves, often there is a combination of muscular rheumatism. A severe attack prevents

sleep and the general health suffers. The exciting cause is generally cold, or accumulations of fæcal matter, but anæmia, rheumatic or gouty conditions, chronic syphilis, and alcoholism predispose. *Treatment.*—Predisposing conditions if present must be treated. If not present, colchicum with alkalies, warmth to the parts, hypodermic injections of morphia, fly blisters, galvanism, punctures with a fine needle, hot alkaline baths, massage.

Scrofula consists of deposit of tubercle (p. 619) in various internal structures, particularly the glands, probably in consequence of their being least organised. As the tubercle cannot be discharged as it is from the lungs in phthisis, it assumes a characteristic cheesy appearance, and becomes mixed with pus. Scrofula is hereditary especially from syphilitic progenitors, but whatever lessens health and strength tends to excite scrofula, or may even originate it. For example, Mussulman perdah women, whose time is spent in close ill-ventilated rooms, especially if in large cities, are particularly liable to scrofula and phthisis. *Treatment* is *preventive*, by avoiding the predisposing and exciting causes; *curative*, by carrying out the rules of health, and treating the multitudinous local manifestations as they arise, on established medical or surgical principles.

Sea Sickness.—Liability depends on peculiarities of constitution. Numerous remedies have been proposed, but none are of much use except for a voyage of a few hours, when a tight belt round the bowels, or an ice-bag to the spine may prove preventive. The best means of preventing sea-sickness are previously to commencing a voyage taking several purgative doses, followed by small doses of bromide of sodium, or if this cannot be done, the removal of acidity by an emetic composed of a tea-spoonful of mustard immediately before embarking.

Septicæmia has been distinguished from pyæmia, as having no necessary connection with any local process, and because the poison producing it has no tendency to multiply in the organism. Although the system may be saturated with poison the subject dies without any secondary centres of disease. When septicæmia is diagnosed the only treatment is supporting the strength.

Spermatorrhœa is not common except as an occasional nocturnal discharge. Glairy, clear, or ropy discharges from the urethra, or from the prostate gland, are often mistaken by patients for spermatorrhœa, an error which certain quacks for

their own advantage foster. The recurrence of spermatorrhœa weakens the system and depresses the patient. *Treatment.*—Change of scene, aperients, going to the closet in the evening, a hard bed, light covering; patient should not lie on the back; tonics, especially iron and quinine bromides.

Small-pox.—Incubation, twelve days. *Cause.*—A specific poison. *Symptoms.*—Shivering, malaise, pain in back, often vomiting succeeded by fever. On the second or third day papular eruption, first on forehead, neck, hands, becoming vesicular on the fifth day, then pustular with umbilication of the centre. About the ninth day the pustules break with peculiar smell, and scabs form, which fall off about the fourteenth day, leaving a red stain; or, if the true skin is implicated, permanent marks. Fever abates when the eruption appears, and increases as it matures. Varieties: *discreta*, *confluens*, the latter often attended with implication of mouth and throat, dyspnœa, and cough. *Treatment.*—Aperients, well-ventilated room, isolation, light nourishing diet; if severe, secondary fever stimulants. Bromide or chloral for restlessness, cold cream or carbolic oil to the pustules. Prevent scratching. Warm baths during decrustation.

Skin Diseases.—In addition to those treated of as common in India, the following require notice: 1. Rashes. A. *Erythema.* A non-infective superficial inflammation of the skin, the red hue of which disappears temporarily on pressure, attended with slight itching or burning. *Symptomatic*, *idiopathic*, and *erythema nodosum* are the divisions; the latter occurring on the legs chiefly of young girls. *Treatment.*—Soothing applications and tonics. B. *Roseola*, *rose rash*, *tooth rash*, *red gum.* Bright spots a little elevated, sometimes accompanied by fever, may assume the form of rosy rings (*annulata*). Distinguished from measles by its occurring suddenly without catarrhal symptoms, and by the rash having no regular site; from scarlet fever by the absence of sore throat; from erythema by its more rosy tint. Alteratives, laxatives, tonics, lancing the gums if swollen. C. *Urticaria*: *nettle rash.* Long white wheals surrounded by red margin appearing suddenly, accompanied by itching, and often by vomiting. Frequently follows certain articles of food, or drinking cold water when the body is heated. In other cases it depends on digestive derangements. If the stomach contains indigestible food, pro-

mote vomiting by a mustard emetic. In other cases aperients. Itching is alleviated by sal volatile and water.

2. Vesicles. A. *Eczema. Symptoms.*—Redness, slight swelling, papulation, vesiculation, exudation, incrustation, desquamation, thickening, hardening, fissure or chapping, accompanied by burning pain and intolerable itching. Upwards of twenty varieties described, but they are merely different phases. Everything giving rise to hyperæmia of the skin will in some constitutions excite eczema. Constitutionally the cause may be traced to disordered digestion, dentition, painful menstruation, pregnancy, nervous disturbances. *Treatment* is therefore constitutional and local ; the former with reference to any error recognised ; the latter soothing in the acute, stimulant in the chronic forms. B. *Herpes.* Varieties : *labialis, præputialis, zoster iris.* In the first there is a patch of inflamed vesicles. In *zoster*, *zona*, or shingle, there is a belt of vesicles round half the body. Herpes iris occurs on the forehead in connection with neuralgia. The vesicles cause burning, tingling, smarting, and on the forehead stinging pain, often severe, and where a mark may be left. *Causes* not well-defined. *Treatment.*—Zinc lotion, lime water, anodyne applications ; aperients and salines. C. *Pemphigus*, *pompholyx*, or *blebs.* Bullæ containing serum form on the skin, burst, dry up, and produce crusts. Most frequent in teething children. May occur without any assignable cause, except general bad health. *Treatment.*—Tonics, puncturing the fully formed bullæ, and dressing with zinc or tar ointment. D. *Prurigo*, or *pruritus.* Itching of some part of the skin, occurring generally in old people, and neurotic in its nature. *Locality.*—It affects the anus and clitoris, the genital organs of women, especially in diabetes, but it may occur all over the body (*prurigo formicans*). Small microscopical vesicles first form, and scratching leads to irritating sores and scabs. Cleanliness, sponging with hot poppy-water, sulphur ointment, tonics, arsenic especially, bromide and choral to procure rest. A washleather cover to prevent irritation from clothing. E. *Sudamina*, or *miliaria*, generally associated with profuse sweating, and occurring during most fevers. The vesicles are as large as millet seeds, and appear irregularly. The hyperæmic base on which they develope gives them a red appearance (*M. rubra*), afterwards the serum becomes opaque and the redness declines (*M. alba*). They subside and

dry into minute scales. Sponging with vinegar and water the best application to mitigate itching; otherwise nothing required.

3. Pustules. A. *Acne*, or *copper nose*. Inflamed hair-follicles, appearing usually on the face. Varieties: *rosacea punctata*, *conformis*, *pustulosa*, *indurata*. But all are simply phases of the inflammatory process, when confined to the chin, termed *sycosis* or *chinwelk*. In bad cases the beard falls off. *Treatment.*—Good hygiene, attention to bowels, tonics. When on the chin, hair should be cut close, then poultices till the surface is clean, afterwards carbolic acid ointment. B. *Favus*, *porrigo favosa*, *crusted ringworm*. Commences in the same manner as ringworm of the scalp, but as the eruption spreads it is not circular. The vesicles soon become pustular, and the secretion dying on the surface assumes the appearance of light, sulphur coloured, circular cupped crusts, penetrated by hairs. Tumefaction, redness, soreness, and baldness result. The crusts emit a characteristic mouse-like odour, caused by a fungus (*Achorion Schönleinii*). The favi must be removed by poulticing, then sulphurous acid lotion, or carbolic acid ointment. C. *Impetigo*, called also *pustular eczema*. Commences as a vesicle which soon becomes pustular, often on the face, but may appear anywhere. There are successive crops, eventually terminating in scabs, said to be contagious. Attention to general health; *unguentum Hydrargyri ammoniati dilute*, to the red surface beneath the scabs. D. *Rupia*. Generally syphilitic. Bullæ form, containing serum at first, and afterwards pus. A thick black scab then forms, beneath which ulceration progresses, and the incrustation assumes a stratified or conical appearance. Good diet, iodide, arsenic required. Locally antiseptic dressings, or iodoform.

4. Scales. *Psoriasis*, or *dry tetter*. Varieties named according to appearance, or according to the part it presents on. One form begins as small round, shining, itching, circular spots, with thin white scales. This circular form is sometimes called *lepra*, and may at first be mistaken for ringworm, but psoriasis is scaly, which ringworm is not. There are several or many patches of psoriasis, while ringworm is generally single. The circular patches, spreading many inches, become broken at the margin, while the interior skin, after being reddened, resumes its natural appearance. A second form commences as irregularly-shaped patches, chiefly

in the flexures of the limbs. When the palms of the hands are affected (*psoriasis palmaris*) it has been confounded with eczema and termed 'Grocer's itch.' A third form is known as *pityriasis*, differing in showing much smaller scales and in being more diffused, occasionally over the whole body. *Causes.*—Not well understood; sometimes seems to depend on digestive disorders, which it may accompany. *Treatment.*—Attention to diet, arsenic. Itching may be relieved by opium lotion; equal parts of tar, spirits of wine, and soft soap is a favourite application. Mercurial ointment often does good. In chronic psoriasis sulphur baths.

Spinal Cord, Diseases of.—1. *Anæmia*; 2. *Hyperæmia*; 3. *Hæmorrhage*; 4. *Inflammation*; 5. *Inflammation of the membranes*, have been described, but there is little certain known as to symptoms, which have been enumerated chiefly from a consideration of what occurs after accident or after exposure to the sun (p. 367). Hæmorrhages, however, are known to occur not only from violence, but also from a pre-existing morbid condition as tumour, or more rarely as a primary event, especially in scorbutic states. Hæmorrhage generally presents in the grey matter, and the symptoms, both of hæmorrhage and of the other conditions mentioned, vary with situation. There may be severe pain increased by movement, sensation as of a cord round the body, motor and sensory paralyses, complete or incomplete, of the lower extremities, bladder, and rectum; or hemiplegia if the disease is limited to the grey matter of one-half the cord. *Treatment.*—Quiet, rest, ice to the spine, attention to the bladder. 6. *Locomotor ataxy* (p. 609). 7. *Progressive muscular atrophy* (p. 621). 8. *Spinal paralysis acute* is now regarded as the same disease as infantile paralysis. The morbid changes are believed to consist of atrophy of the anterior cornua and their nerve cells, and of the anterior columns in the cervical and lumbar enlargements. Exciting causes often appear to be cold, wet, injury. Paralysis is sudden, without loss of sensibility, but in children it may commence with convulsions. The leg or legs are mostly affected, and they rapidly waste. After days or weeks the loss of power gradually disappears, but it may leave in children club-foot or other deformities. *Treatment.*—Strychnine, cod liver oil, arsenic, phosphorus, good diet and hygiene, shampooing, friction, warmth. 9. *Spinal paralysis, acute ascending*, has been observed

as advancing rapidly from below upwards, so as finally to implicate the parts depending on the medulla oblongata. *Causes* and *treatment* equally obscure. 10. *Spinal paralysis, chronic atrophic*, is a slow form of the acute spinal paralysis, ascending or descending. 11. *Spinal paralysis, spasmodic*, depends on fatty degeneration and sclerosis of the spinal cord (*vide* Softening), and the symptoms do not differ materially from those of locomotor ataxy, while the treatment is the same. 12. *Sclerosis* of the cord depends on overgrowth of neuroglia, or that fibroid overgrowth which forms the basis of so many examples of cirrhosis or sclerosis in different organs and tissues, being a process pathologically intermediate between inflammation and degeneration. Sclerosis may cause *paralysis agitans* (p. 615), and is generally associated with softening, and the symptoms and treatment do not materially differ. 13. *Spinal irritation*, or *rachialgia*, chiefly occurs to young girls, and no organic alteration has been discovered. The symptoms are multitudinous, and chiefly of an hysterical character, from catalepsy and spasms, tonic or clonic, to functional disorders of all organs ; but associated with more or less hysterical pain in some part of the spine. It is, in fact, hysteria, and should be treated as such. 14. *Softening* consists essentially of a fatty degeneration of nerve fibres, and is often combined with fibroid interstitial overgrowth, so that patches of sclerosis are blended with the softening. It has been attributed, without any good grounds, to anæmia, hyperæmia, hæmorrhage, and inflammation. It may involve the whole thickness of any part of the cord (transverse softening) or only parts (circumscribed). *Symptoms*, therefore, are exceedingly diverse. In complete transverse softening, at the mid-dorsal region, the most usual seat, the motor paralysis of the legs and abdominal muscles is absolute, the sensibility is lost, and there is no reflex action. Incontinence of urine and fæces ; bed sores ; and death from exhaustion. *Treatment.*—Palliative.

Stomach, Diseases of.—1. *Albuminoid, waxy*, or *lardaceous disease* may affect the stomach, but it is associated with similar disease in other organs, and there are no special symptoms. 2. *Atony* of the stomach is described, but it is a part of atonic dyspepsia (*vide* p. 228). 3. *Atrophy* is marked by anæmia, and is only to be diagnosed by the exclusion of all other causes which produce anæmia, and it is questionable if it ever occurs as a dis-

tinct disease. 4. *Cancer; scirrhus* is the most common form. *Symptoms* insidious, commencing with ordinary dyspeptic manifestations. Pain at epigastrium of a dull gnawing nature, increased during digestion, but present when the stomach is empty. Vomiting, loss of appetite, emaciation, slight jaundice, gastric tumour hard and nodular, usually fixed by adhesions, and generally at the epigastrium, as the pylorus is chiefly affected. The diagnosis is principally between cancer and ulcer. The pain is more severe, more influenced by food, more relieved by vomiting in ulcer than cancer. In cancer it is less fixed to one spot, and more neuralgic. Severe hæmatemesis is characteristic of ulcer, frequent coffee-ground vomit of cancer. Cancer seldom appears under thirty-five. The presence of a tumour is decisive. *Treatment* can only be palliative. 5, 6. Both *contraction* and *dilatation* of the stomach are described, but they are rare, and cancer affecting the pylorus is the usual cause of the latter. 7. *Fibroid thickening* of the stomach is another malady which cannot be accurately diagnosed. 8. *Inflammation*, or *gastritis*, may result from the latter condition, but two special forms are recognised, viz., *catarrhal gastritis*, and *erythematous gastritis*. Gastritis occurs to gouty and rheumatic subjects, and is relieved when the primary affection appears. It is also consequent on alcoholism, and presents in those suffering from hepatic cirrhosis and cardiac disease. Gastritis is usually an effect of remittent, typhoid, and other fevers; but it also occurs as the result of dyspepsia (pp. 223, 292). 9. *Chronic gastritis* is also a symptom of the various forms of dyspepsia, however caused (p. 220). 10. *Ulcer* is marked by pain, increased by food, relieved by bending forward, usually referred to the epigastrium or back; often tenderness; vomiting a variable symptom; appetite seldom affected, but patients do not eat freely, from dread of pain. Ulcers are most common in the pyloric region, and may give rise to *stricture of the pylorus* by causing spasm of the pyloric muscular fibres. Ulcers are most met with in young people and females, becoming again more prevalent in advanced age. They may arise from sloughing of a portion of the mucous membrane from the action of the gastric juice when abnormally acid, or from the alkalinity of the coats of the stomach being abnormally diminished; from general anæmia, however caused; from the death of small portions of the membrane; from embolism of arteries. They have

also been attributed to catarrh, and in old people to diseased arteries; also to fatty and fibroid degeneration. *Treatment.*—*Vide* Hæmatemesis. 11. *Perforation* may result from any form of ulceration, but simple ulcers are more prone to penetrate than cancerous, in which adhesions are more likely to form. Perforation often takes place with startling suddenness, and is attended by severe pain, retching, and vomiting, with tendency to collapse. No treatment is of much avail, but recovery has apparently occurred.

Trichina Spiralis occurs in the muscular tissue as a minute worm coiled in an oval cyst, derived chiefly from pork. The cysts are dissolved by the gastric juice, and the parasites set free, which then find their way into the body. *Symptoms.*—Intestinal disturbance simulating typhoid, muscular lameness, muscular pains like rheumatism, and stiffness. In eight or ten days œdema of face without albumen, general prostration, insomnia, profuse sweating. Symptoms only can be treated.

Uræmia.—When there is albumen in the urine, although the fact may not be known, uræmia may commence insidiously with headache, imperfections of vision, drowsiness, followed by convulsive paroxysms, insensibility, stertorous breathing, pale face, dilated pupils, permanent depression of temperature. The retina may show albuminuric patches in the shape of white deposits. Such cases may be mistaken for apoplexy (p. 577), supposed to be due to circulation of urea in the blood, or to the conversion of urea into carbonate of ammonia, or, thirdly, to the accumulation in the blood of the first products of tissue change (*creatine*, creatinine, &c.), which are normally converted in the kidneys into urea and uric acid. There is no anatomical change in the nervous centres. *Treatment.*—Benzoic acid as a preventive, which is believed to convert the poisonous alkaloid into a harmless salt; cathartics, sudorifics. When an uræmic attack has commenced little can be done. Withdrawal of ascitic fluid, and subcutaneous injection of digitalin have succeeded. In puerperal convulsions from uræmia, chloral (p. 622).

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